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THERAPEUSIS OF INTERNAL DISEASES

MT. F.

THERAPEUSIS OF INTERNAL DISEASES

EDITED BY

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VOLUME IV

NEW YORK AND LONDON
D. APPLETON AND COMPANY
1913

129390
24/9/13

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Printed in the United States of America

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THERAPEUSIS OF INTERNAL DISEASES

VOLUME IV

I. DISEASES OF THE KIDNEYS

CHAPTER I

SYMPTOMS

F. Forchheimer

MOVABLE KIDNEY

(*Enteroptosis*)

Movable organs are not uncommon; Hector Mackenzie examined 2,801 females of all ages and found movable kidneys in 516. In 1,607 males a movable kidney was found only 11 times. These statements establish the fact, already known, that enteroptosis is much more common in females than in males. The cause of this is easily understood by considering the different conditions which exist in the male and female that make for the support of the abdominal organs. The uterus and adnexa, originally in a horizontal position, now have the same supports for an upright position of the body. During the process of evolution the supports have not changed, so that, in the female, there is weakness of support for the pelvis and indirectly for the abdominal organs. The abdominal organs are held in place by the abdominal walls, and all those conditions, childbirth, tight lacing, etc., which weaken them may and do cause this condition. There is intraabdominal support, as well; this is accounted for by the intraabdominal fat; for the kidneys especially there is a thick layer of fat surrounding them. Intraabdominal vascular tonus should also be considered; the more it is diminished the less support there is to the intraabdominal organs. Indirectly this affects the kidney. It is thoroughly recognized that frequently two, or even more, organs are out of place at the same time when the external supports are normal. This can only be due to some intraabdominal cause, and when the fat is normal the circulatory condition accounts for it. Anything acting mechanically, such as strain in lifting, traumatism, or increase in weight of the kidney, may be followed by dislocation of the kidney; literally the kidney is dislocated, but it is doubtful if this condition should be considered with that of enteroptosis. Certainly, for therapeutic purposes, this is not the case.

In treating these patients the physician should be careful how he communicates the fact that there is a movable or floating kidney. If, as seems to be the case, about 20 per cent. of these patients do not know that the kidney is displaced (Hector Mackenzie, l. c.), it is well for the physician to study his patient before he makes the communication. Many a patient has been made an invalid by failure to follow this rule; neurasthenic, hypochondriacal, and sometimes psychotic. The treatment should be conducted along causal indications. It is not necessary to emphasize the fact that, when there is no weakness in the external abdominal supports, a bandage is only of symtomatic value. When there is this weakness a bandage should always be applied. The principal requirements for a good bandage are that it be supported by the most rigid base that can be found, in this disease the ribs and the pelvis; that it be sufficiently rigid itself to form a good support for the pad (or pads) that must be added to hold up the organ (or organs); and, finally, that it move not too much or too little—too much in that insufficient support is given; too little in that respiratory movements and the necessary movements of the body be interfered with. The designing, fitting, and application of the bandage should be under the supervision of a physician who is prepared to undertake such cases.

In the case of tight lacing the cause should be removed; sometimes this is deferred too long and then nothing can be done for the condition except surgical treatment. When the diagnosis is made early enough, and the results of the condition are explained to the patient, many get well without further treatment.

Those cases in which intraabdominal support is at fault do well enough when the cause can be removed. Not infrequently in these cases we find a combination of causes, flaccid abdominal walls and lack of fat intraabdominally, the first to be treated by the bandage, the second by diet and rest. The object of dieting should be the making of fat; for this purpose a large number of calories are prescribed, which are produced from a mixed diet consisting of large quantities of carbohydrates and fats—the albumins in smaller quantities absolutely and relatively.

The other form of lack of internal support is found in neurasthenics and hysterics, more frequently in the former than in the latter. They should be treated generally (see Neurasthenia and Hysteria). Locally it may be necessary to apply the bandage. These patients should be induced to try gymnastics, abdominal massage, and electricity. Postural treatment seems very valuable in this class of cases. They should be taught to lie upon an inclined plane, the head low and the feet high, beginning with slight and ending with great inclination; at first for a short time and then for a longer time.

The results from this form of treatment are excellent, but, do what we may, there remain a certain number of cases in which a surgical operation, nephrectomy or nephrorrhaphy, should be performed. The indications for operation seem to be, at the present time of writing: when the patient's life is endangered, and when the patient is prevented from pursuing his occupation or enjoying his life. The first occurs in the floating kidney, in which there is hydronephrosis; when there is no relief in frequent attacks of Dietl's crises, either nephrectomy or nephrorrhaphy are indicated. Hahn's operation, nephrorrhaphy, should always be preferred on account of its comparative lack of danger.

ANURIA

This symptom should be treated as to its causes. For its treatment in nephritis the reader is referred to the chapter on Bright's disease. The obstructive form of anuria calls for surgical treatment. In the nonobstructive form the cause should be sought for, and, if possible, removed, which is rarely possible. The damage done to the kidneys is possibly only temporary, and it is our duty to facilitate the return of the normal condition, at the same time seeing that no harm occurs to the patient while this is going on. For this purpose diuretics should not be prescribed immediately, as it is not certain how much good or harm we may do in administering them. Especially is this the case in the toxic forms. It is much better to try to relieve the kidney by giving sudorifics or cathartics and functional insufficiency should not be embarrassed by the introduction of abnormally large quantities of water. Dry cups to the loins, poultices, and hot applications are valuable. In these circumstances normal saline solution should not be employed, as NaCl is said to irritate the kidneys.

On the other hand, when the kidneys are normal, NaCl in normal (0.60 per cent.) solution, either by hypodermoclysis or by intravenous injection, should be employed. Hot rectal injections are most valuable in this form, which is found in collapse in cholera, after operations; indeed, in any condition.

There is little to be said about hysterical anuria. Its treatment is most difficult; nephrotomy has been performed with no immediately beneficial result (see Hysteria).

HEMATURIA

The causes of this symptom are treated in connection with the diseases in which it occurs. It will therefore be considered simply as a symptom which should be controlled. The patient should always be put to bed and kept there at least until macroscopic blood has disappeared from the urine. His diet should be unirritating and should contain nothing which may irritate the kidney. When the underlying cause is known, this may not be necessary. At all events, nothing should be given which causes the urine to become irritating. Hemostatics should be administered unless causal treatment can be applied immediately. The remedies which may be employed are (and I give them in the order in which they have been most serviceable to me): adrenalin, calcium lactate, ergot. Pituitin should be considered because of its excellent tonic effect upon blood vessels. The other hemostatics should be employed only in milder cases, and in these they have seemed to be useful. I refer especially to tannic and gallic acids and lead acetate. In addition, large doses of hexamethylenamin should be given in order to prevent possible infection of the urinary tract.

Nephrotomy should be considered when no cause can be found and the bleeding does not cease. It does not always help matters by checking the hemorrhage; it may, however, be life-saving when the bleeding is checked by it, and, moreover, the cause of the trouble may be found.

HEMOGLOBINURIA

Toxic Hemoglobinuria.—Methemoglobinuria and hematoporphyrinuria are included in our present topic. Hemoglobinuria is found in connection with infectious diseases: syphilis, malaria, scarlatina, typhus, typhoid fever, and yellow fever. It may be the result of purpura or scurvy. It is also caused by a number of intoxications; with acids, such as sulphuric or hydrochloric (chronic intoxication), by the ingestion of solanin (sprouting potatoes), by inhalation of hydrogen arsenic, by the ingestion of uncooked mushrooms, and of soap bark (saponin). In all these conditions there is hemolysis, as it also is in *methemoglobinuria.* This subject has become more and more important as the number of causes has increased, largely on account of the introduction of drugs which produce methemoglobinuria. The clinical picture which existed before the era of new drugs was that of potassium chlorate intoxication, usually fatal but rare. With the introduction of drugs derived from coal tar, methemoglobinuria has become rather common, and mild cases are recognized, which recover, as a rule. Acetanilid, phenacetin, antipyrin, pyridin, naphthol, pyrogallic acid and carbolic acid and their various combinations are the principal toxic drugs.

Hematoporphyrinuria most frequently follows the administration of sulphonal or trional.

It will be seen, therefore, that prophylactic treatment is most important in all these conditions. For hemoglobinuria, everything that tends to reduce the toxins, whether it be antitoxic or antibacillary treatment; should act prophylactically. In other words, specific treatment may prevent this form of blood condition. The trades intoxications can be prevented beyond a doubt; it only wants the will, the way is easily found. Sprouting potatoes should never be eaten.

The prevention of poisoning by drugs which produce methemoglobinuria and hematoporphyrinuria is a serious question, as the intoxication may follow medicinal doses prescribed by the physician. The taking of these drugs habitually and in large doses without the advice of a physician cannot be prevented and does not concern the physician except when he is called to advise treatment. When, however, a physician prescribes a drug and toxic effects follow, although he has done all that should be done, he is blamed for the result. This is perfectly natural, as the layman sees only the direct cause and not the contributing ones which make for the bad result. It is well, then, that the physician exercise great care in prescribing drugs of the class under consideration. Upon the whole it may be said that the antipyrin group gives less trouble than the acetanilid group. The further the derivatives are developed from the mother substance the milder the effects as a whole. In the present state of drug production it is exceedingly difficult to choose a drug. That one which is recommended by good and, apparently, respectable authority frequently turns out to be very toxic. Physicians not uncommonly feel that new drugs represent advance, at all events that they should prescribe them. The rule should be followed here, as elsewhere, that when a drug fills all the requirements necessary to make it efficacious and safe it should be retained by the practitioner. If he changes to the newer drug he may be changing a bird in the hand for one in the bush, and, what is worse, discarding a tool which fits him perfectly and which he has learned to use as one only can an old tool.

Treatment —In methemoglobinuria the drug which causes the intoxication should be immediately discontinued. In the acute form of intoxication the treatment can only be symptomatic. Oxygen inhalations are recommended; they may be valuable as not only the hemoglobin but the plasma of the blood as well carries oxygen for its internal respiration. Intravenous injection of normal saline solution should be tried in all the bad cases; among other effects, it dilutes the toxic substances in the blood, producing temporary amelioration which may be sufficient to turn the tide in favor of the patient. Even Sahli's lavage of the blood may be tried. In some instances cardiac remedies are required; caffein seems to be the most frequently used, and should be given hypodermically. It is not rational to believe that when caffein is given with the coal tar product the cardiac symptoms do not develop. Especially is this true of acetanilid, in which the heart is not so much affected as the respiration. The collapse requires stimulants, camphor, ether hypodermically. The irritation of the kidneys, which comes on early and which may last for some time, should also be treated.

In hematoporphyrinuria the treatment consists in the removal of the cause, i. e., the drug producing it should be removed, and this is sufficient in the majority of cases. But enough cases of poisoning with sulphonal and trional are now on record so that we are better able to meet this emergency than formerly. Sulphonal produces by far the worst forms and the most dangerous; trional poisoning is milder and not so often followed by hematoporphyrinuria. In all these forms of hemoglobinuria there are both acute and chronic forms.

PAROXYSMAL HEMOGLOBINURIA

It is difficult to discuss the treatment of a disease in which so little is known of its cause. As for *prophylaxis* there is more chance for success, as by careful study of the individual case the immediate cause, i. e., the cause of the attack, may be found out. In one case which came under my observation I could not find any cause for the classical attack of paroxysmal hemoglobinuria. It is known that refrigeration brings on an attack in some patients, probably the largest number are produced in this way—cold applied to the body as a whole, exposure to wet and cold—and experimentally by taking a very cold bath. Even local application of cold, as a foot bath in ice-cold water, may cause an attack in those predisposed to this disease. Again there are patients in whom anxiety, worry, exertion are followed by an attack. In predisposed women menstruation sometimes causes a paroxysm. Except in the latter case, prevention is simple. Either the patient must be very careful to protect himself against cold, sometimes impossible of accomplishment, or he should move to a warm climate. Before doing this it is necessary to be absolutely sure that no attacks occur except as the result of cold.

Treatment.—For the treatment the fact must be considered that, in a number of cases, syphilis or a syphilitic history was found, and those who report these cases state that the condition, evidently the cause or a cause, had been removed by antisyphilitic treatment. So much so has this been the case and the therapeutic results so good that the advice is given to put all patients with paroxysmal hemoglobinuria through a course of antisyphilitic treatment. The connection with syphilis can be determined by the existence of a positive Wassermann reaction, so that when this exists active antisyphilitic treatment should be instituted. Even when there is no syphilitic history antisyphilitic treatment has been employed with good results. The employment of the Wassermann test should, therefore, be recommended in all these patients for the purpose of discovering the etiology in the individual case.

There exists an intermittent form of hemoglobinuria which is due to an indirect effect of malaria. The hemoglobinuria which exists in malaria may become intermittent and then resemble the paroxysmal form. In these cases it is well to begin with a course of arsenic, to be followed by quinin. If, for one reason or another, the patient requires prompt relief, quinin must be given as described in the chapter on malaria. We must never forget that in predisposed people quinin may produce hemoglobinuria, and arsenic seems to prevent it.

During the attack the patient must be kept in bed. The diet should be bland and especially unirritating to the kidneys (v. Nephritis). A large quantity of fluid should be taken by the patient so that the hemoglobin may be worked out of the kidneys. This should be done in every case unless there is a possibility of doing harm with it.

Inhalations of nitrite of amyl have been recommended in order to abort these attacks; they may be tried when there is no contraindication.

The mortality in this condition is very small, although fatal cases are recorded; some patients go about their business unless the urine is very bad and some continue under any condition.

OXALURIA

For many years a so-called oxalic acid diathesis was accepted; indeed, is still accepted. While oxalates are found in the urine in certain combinations of symptoms, it by no means follows that they cause the symptoms. If we look at the origin of oxalic acid and its salts we find that various views exist: one, in which the substance is supposed to be exogenic; the other, in which it is considered endogenic; and a third, in which both are considered as playing a rôle. It is probable that the latter view is correct. The greater part of the oxalic acid is derived from the food, the lesser from metabolic changes which have not been definitely settled. It is claimed by some that in the endogenous form oxalates are the result of albuminous metabolism, by others of changes in the carbohydrate group. Whichever it may be, it is certain that the oxalates can be reduced most readily by excluding foods which contain much oxalic acid.

The combination of symptoms which were supposed to be due to oxaluria are those of chronic intestinal intoxication: symptoms on the part of the gastrointestinal tract, the nervous system, the urinary organs. Physical examination reveals the changes in the intestine which are found in chronic intestinal autointoxication. The urinalysis shows increase in the indican, with or without increase of the aromatic sulphates, and of calcium oxalate crystals, all found with more or less regularity in chronic intestinal autointoxication. When we study the question from this point of view, it is not likely that the origin of the symptoms is due to the oxalates. Moreover, all the symptoms which are ascribed to oxaluria are found in chronic autointoxication of the intestinal type, without the presence of oxalates in the urine. It seems more than likely, then, that we have confused the effects with the cause, and when oxaluria per se requires treatment its correct treatment is that of chronic intestinal autointoxication.

It is especially the local effect of the so-called oxalic diathesis which requires treatment. The urinary evidences of this condition are the constant presence of blood in the urine in microscopic or macroscopic quantities, and of calcium oxalate crystals, which affect the patient generally as the result of hematuria or locally by irritating the urinary passages or producing calculi. It is for these reasons that oxaluria requires especial therapeutic mention. The first problem is to reduce the quantity in the urine. For this purpose it is necessary to restrict the diet in such a way that oxalic acid is not introduced into the economy. It must not, however, be done with the idea that oxalates can be removed entirely from the urine, because, as has been stated before, the oxalates are metabolic end products.

The first measure to be enforced is *diet,* which must not include articles which are known to contain large quantities of calcium oxalate: rhubarb, tomatoes, pineapple, apples, sorrel, strawberries, and lemons should be eaten sparingly or refrained from altogether in the beginning of the treatment. It is impossible to prevent the introduction of oxalate of lime, as it is practically found in all vegetables. Excess in eating should be forbidden. The stomach should be treated; it is an accepted fact that there is a connection between oxalic acid formation and dyspepsia, usually due to subacidity. Much good may be done, in this condition, by the internal administration of mineral acids: dilute hydrochloric or dilute nitrohydrochloric acids (0.6-1.0 c. c., m xv), given well diluted and after meals. As oxalic acid is formed from uric acid, the treatment of gout may be applied with advantage in many instances in which there is a history of gout. In those cases which are of the dyspeptic or gouty type, which, as a rule, if not always, is due to chronic intestinal autointoxication, cures such as are conducted in Karlsbad and in places having sulphur waters are valuable and successful. (See Vol. I, Sec. I, Chapter IV.)

PHOSPHATURIA

The origin of the phosphoric acid in the urine is from two sources: it is exogenous or endogenous. By far the larger amount comes from the food, and for our present purpose need not be considered. This is the case except in neurotic subjects who watch their urine and are guided in their feelings by the presence or absence of its phosphatic precipitate. In these patients it is well to explain to them how the amount of the phosphates is determined, and that their test is of no value whatsoever as the precipitation of the phosphates in the urine depends upon many factors. Endogenously phosphoric acid is formed from organic combinations which are especially found in the nervous system: nuclein, glycero- and oleophosphoric acids, lecithin, and protagon. Under these circumstances it is not strange that the purin bodies are usually increased in this form of phosphaturia.

Under all circumstances the diagnosis of phosphaturia should only be made after qualitative and quantitative analyses are done, otherwise the subject becomes one of those general terms which cover over poor diagnoses and do much harm. When the diagnosis has been properly made, much can be done by treatment. It is necessary that patients who present too much phosphoric acid in the urine, followed by disturbances that can be attributed to it, should be treated. The diet should be arranged so that albumin is taken in minimum quantities, carbohydrates making up the deficit in calories. Moreover, vegetable albumin and milk may be given. In the purely endogenous form diet does not seem very valuable. The general condition, reduction in wear and tear, curtailment or abstinence from work, should be recommended. In these cases the treatment applied to all reduced neurotics should be applied, as to food, rest, and general measures.

It has been shown by von Noorden and his school that the administration of calcium carbonate prevents the phosphates from being eliminated by the urine. Elimination takes place under these circumstances into the bowels at all times, and at least one-half and more of the phosphates can be prevented from leaving the system by the kidneys. Creta preparata (gm. 1-2—gr. xv-xxx) is given twice or three times a day. There is no difficulty in verifying this statement, and, in as far as preventing the development of local conditions in the kidneys and bladder is concerned, this measure may be valuable. As a rule, however, more is gained by preventing the formation of phosphates than by removing them.

REFERENCE

Mackenzie, Hector. Jour. A. M. A., 5, 1907.

CHAPTER II

NEPHRITIS

Joseph L. Miller

INTRODUCTION

Before undertaking the discussion of the therapeutics of nephritis, it has seemed advisable to discuss in more or less detail the normal and pathological physiology of the kidney. Therapeutics is largely an effort to convert pathological into normal physiology. In the past the pharmacologist has confined his studies largely to the action of drugs on normal animals, and our drug therapeutics is therefore founded largely upon the action of drugs on normal animals. In recent years experimental pathology and experimental therapeutics have attempted, with excellent success, to fill in the gap. Through their work the field of pathological physiology has been greatly extended. They have also furnished us with much-needed information in regard to the action of drugs in pathological conditions—the only state that requires therapeutic measures. Intelligent therapeutics and pathological physiology should go hand in hand. The kidney is an organ which offers special facilities for physiological investigation. It is for this reason that pathological physiology occupies so much space in the discussion of the therapeutics of nephritis.

NORMAL PHYSIOLOGY OF THE KIDNEY

Theories of Renal Secretion.—The physiology of urinary secretion has not been definitely determined. Two theories, each with worthy defenders, must be presented. Ludwig's theory maintains that we have in the secretion of urine a simple process of filtration with the glomerulus acting as the filter eliminating from the blood not merely the water but also the solid constituents of the urine, viz., the inorganic salts and specific elements—urea, etc. To explain the discrepancy between the proportion of the various inorganic salts and especially the urea, in the circulating blood and urine, he assumes that absorption of water and varying amounts of the other constituents takes place in the passage of the glomerular filtrate through the uriniferous tubules. In recent years the adherents of Ludwig's theory have introduced some modifications, claiming now that the absorption taking place in the convoluted tubules is selective in character, so that the theory as now held is not based upon the purely mechanical process of filtration and diffusion. Certain anatomical conditions in the kidney lend themselves to this mechanical theory. The efferent vessel to the glomerules is smaller than the afferent, and for this reason the pressure in the capillary tuft would be higher than in ordinary capillaries. The great length of the urinary tubules might be considered as especially adapted for the reabsorption of the fluid passing through them, but as Stewart states such anatomical evidence is not especially convincing, as the absorption of water is not a function of the seminal tubules, which may be 60 to 70 cm. in length. Furthermore, the physical laws of filtration, diffusion, and absorption, as now understood, cannot account for the process. Serum albumin and egg albumin are both indiffusible through animal membranes, yet one passes through the glomerulus without escaping into the urine, the other is promptly eliminated. Two diffusible substances, as sugar and urea, are present together in the circulating blood. The latter is eliminated as a waste product, the former retained. If, however, the amount of sugar in the blood passes a certain limit, more than three parts per 1,000, it is promptly excreted. With a pure filtration theory it is difficult to explain how, in a starving animal, the amount of inorganic substance in the urine may be greatly reduced, while the proportional amount in the blood is only slightly changed. At the present time the vital theory of Bowman has the stronger support, permitting of a selective action of the cells. This theory assumes that the glomerular epithelium removes from the blood the water and salt of the urine by an act of secretion. The urinary tubules are not primarily for absorption, but their epithelial cells are actively secretory, withdrawing from the blood especially the organic constituent known as urea, although Howell states that the precise secretory products of glomerulus and tubule are not clearly defined by the adherents of the vital theory.

Secretory nerves for the kidney have not been demonstrated, the secretion being directly dependent upon the rate of blood flow. When the arterial pressure falls below 40 mm. of mercury as following shock, hemorrhage or extreme impairment of the heart muscle, the secretion of urine ceases. Everything which increases the blood flow, as increased blood pressure or local dilatation of the renal vessels, without reduction in the general blood pressure, such, for instance, as follows the use of caffein, intensifies the secretion of urine. So far as known, the only way the secretion in the kidney can be affected by the central nervous system is through a local nervous mechanism by means of which the secretory activity is modified by corresponding alterations in the local blood supply.

The kidney is a very vascular organ, and it has been estimated that when under the influence of diuretics, within a minute's time an amount of blood equal to its own weight flows through the kidney. This is from four to nineteen times as great as the average supply of other organs in the systemic circulation.

The Composition of Urine.—The normal urine in man is yellowish in color, with an average specific gravity of 1.020. The reaction is usually acid, until recently attributed chiefly to the acid sodium phosphates. Folin's investigation shows, however, that it is due perhaps largely to organic acids. In addition to the water and inorganic salts the most important constituent of the urine is the urea. It is in this form that almost all of the nitrogen of the body is eliminated, as the amount escaping through the perspiration and feces is comparatively slight. Since nitrogen forms 16 per cent. of the protein molecule, the amount of protein broken down in the body can be determined by multiplying the amount of nitrogen eliminated by 6.25. The daily nitrogen elimination varies, but in an average sized man it would range between 14 and 18 gms., corresponding to 88 and 117 gms. of protein. Of the total nitrogen eliminated, 87.5 per cent., according to Folin, appears in the form of urea. The remaining nitrogen is eliminated in the form of ammonia nitrogen, 4.3 per cent.; creatinin nitrogen, 3.6 per cent. And in the form of purin nitrogen and unknown nitrogen compounds, this latter at times amounts to more than 5 per cent.

Origin and Significance of Urea.—From 30 to 34 gms. of urea is eliminated daily by the normal adult on a mixed diet. As 1 gm. of proteid will yield approximately 1/3 gm. of urea, this would correspond to a proteid destruction of 90 to 100 gms. It has been determined that urea is formed chiefly in the liver. Just the exact steps through which the protein molecule is metabolized to produce urea have not been fully determined. It is well known that when ammonia carbonate is added to the blood and perfused through the liver it is converted into urea. After removal of the liver by Eck fistula, the amount of ammonia in the urine is increased; the urea diminished. Urea then arises from conversion of ammonia salts, this process taking place in the liver. As a result of digestion, proteid material is broken up into its constituent elements and the nitrogen appears chiefly in these forms: ammonia, monamino acids, and diamino bodies. It has not been definitely determined in what form ammonia appears in the blood, but most likely as a carbonate or carbamate. It is probable that a small amount of ammonia may be converted into urea by the action of other tissues than the liver. Kossel and Dakin have demonstrated the presence of a ferment which is capable of splitting up diamino bodies, one of the end products being urea. The urea arising in this manner is formed largely independent of the liver and probably explains the presence of small amounts of urea in the urine after removal of the liver. Finally a certain amount of urea arises from further metabolism of uric acid. In man it is estimated that about one-half the uric acid formed is further changed to urea. The amount of urea nitrogen in the urine varies directly with the amount of proteid ingested, while the other nitrogenous constituents, as creatin, etc., are practically independent of the character of the food, provided it does not contain these bodies when eaten. These nitrogenous compounds are, therefore, formed from the body tissues rather than from the food.

Creatinin next to urea and the ammonia compounds is the most important nitrogenous constituent of the urine. As stated, the amount is independent of the proteid ingested, the daily output varying from 1 to 2 gms. In febrile conditions, where we have increased tissue destruction, the creatinin excretion is increased. The purin bodies, uric acid, xanthin, hypoxanthin, adenin, guanin, also arise chiefly from metabolism of body proteid; only a small amount arising from purin bodies taken as food. Inasmuch as sulphur is found in most proteids its elimination in the urine may be taken as an index of the amount of proteid destruction. Sulphur occurs in three forms, as oxidized inorganic sulphur, conjugate sulphur, and in an unoxidized form as sulphocyanid or ethyl sulphid. Under certain pathological conditions some sulphur may be excreted in an uncombined form.

The chief inorganic salts in the urine are chlorids, sulphates, and phosphates, and arise partly from salts ingested in the food, and partly from the metabolic destruction of body tissues. Of these salts chlorids in the form of sodium chlorid are by far the most abundant, averaging 10 to 15 gms. daily.

Internal Secretion of the Kidneys.—Tigerstedt and Bergman were first to report on the presence of a pressor substance "renin" in kidney extracts. This pressor substance, soluble in normal salt solution, was confined chiefly to the cortical portion of the kidney. It was also soluble in alcohol; non-dialyzable, and destroyed by boiling. Very small amounts, when injected intravenously, caused a rise in the blood pressure, varying from a few mm. to 25 or 30 mm. The following year Lewardsdowsky repeated Tigerstedt's experiment, and, although his results were in a manner confirmatory, he, on the whole, was inclined to discredit Tigerstedt's result. Later Livon, Fiori, and Shaw demonstrated a pressor substance in the kidney, while Vincent and Sheers reported lack of uniformity, at times a rise, again a fall, in blood pressure. Pearce recently repeated these experiments, as he believed the discrepancy in results might be accounted for by kidney extracts of one species being injected into the same or different species. For instance, Tigerstedt used extracts of rabbits' kidney, but at times injected it into a different species. Pearce found that extracts of rabbits' kidney when injected into rabbits and extracts of dogs' kidney when injected into dogs failed to show the presence of a pressor substance, although extracts of rabbits' kidney at times gave a slight rise. Extracts of cats' kidney, when injected into the same species, gave quite uniformly a decided rise in pressure, varying from 22 to 34 mm. An extract of rabbits' kidney injected into a dog gave a fall in pressure. An extract of dogs' kidney, when injected into a rabbit, gave a slight rise in pressure. Pearce has also shown that the urine from a normal dog when injected intravenously into a second dog would cause a marked fall in blood pressure of 40 to 70 mm. Boiling the urine for a few minutes does not destroy this depressor substance, but prolonged heating will cause its disappearance. He was able to precipitate it from the urine by using large amounts of alcohol or ether. Following experimental tubular nephritis, this depressor substance entirely disappeared from the urine, but remains present in the urine of experimental vascular nephritis.

PATHOLOGICAL PHYSIOLOGY OF THE KIDNEYS

The study of the functional disturbance in the kidney has been greatly facilitated by the recent work in experimental nephritis. These studies have given us an opportunity to determine accurately not only the changes in the character of the urine in the vascular and tubular type of nephritis, but also enables us to study accurately the action of various drugs upon the secretion of urine in pathological kidneys. In addition it has thrown considerable light on the causes of edema, anuria, hypertension, heart hypertrophy, and uremia. It has also enabled us to study carefully for the first time the influence of glomerular changes as contrasted with those of the tubules. At the present time we have a large number of nephrotoxic substances, the most important of which are bichlorid of mercury, arsenic, cantharides, chromium, uranium, snake venom, and diphtheria toxins. Some of these act diffusely, affecting both tubule and glomerulus; others are selective in their action. Uranium nitrate, potassium and ammonium chromate, and bichlorid of mercury affect especially the tubular epithelium, attacking slightly, if at all, the glomeruli. Arsenic, cantharides, and snake venom affect chiefly the glomeruli, with very slight action on the tubular epithelium. Diphtheria toxin has a more diffuse action, injuring both glomeruli and tubules. The result of the introduction of any of these substances into a susceptible animal is to produce a particular type of nephritis with appearance of albumin and casts in the urine.

Extensive granular or fatty degeneration of the tubular epithelium with definite necrosis characterizes the epithelial type. In the glomerular form due to arsenic, apparently, there is a paralysis of the capillaries in the tuft, with little if any anatomical changes. However, as shown by physiological methods, serious vascular injury has occurred. Cantharides, on the other hand, produces marked anatomical changes in the glomeruli, desquamation and leukocytic infiltration; and rather marked degenerative changes can also be demonstrated in the convoluted tubules and ascending loop of Henle. Pearce has shown that rattlesnake venom produces still different anatomical changes characterized by extensive exudation and infiltration in both the capsule and glomerular tuft. Tubular involvement is slight or entirely absent.

The study of these various types of nephritis throws some light upon the route through which toxic substances are eliminated from the kidney, as it is probably through the process of elimination that the various degenerative changes are produced. The experimental nephritis described is strictly an acute form. The animal succumbs or complete recovery occurs. Only comparatively recently have we been able to produce experimentally a chronic nephritis. Necrosis of the renal epithelium and some fibrosis were noted by Petroff after prolonged administration of some of the metals. Ophuls later produced a sclerosis of the kidney in dogs and guinea pigs by prolonged feeding of lead carbonate or acetate. He was unable, however, to find at any time albumin or casts in the urine. For this reason it may be questioned whether the condition was actually chronic nephritis. Haven Emerson has produced varying degrees of chronic nephritis by repeated inhalation of alcohol, ether, and chloroform. Uranium nitrate, when given in repeated doses, produces in animals lesions closely resembling the interstitial type of nephritis as seen in man. Even a single acute attack of uranium nephritis may be followed by severe fibrosis, which at times is progressive in character.

As has been stated, experimental nephritis has added not merely to our knowledge of the pathological anatomy of nephritis, but also to the pathological physiology of the kidney as the nature of edema, blood pressure, cardiac hypertrophy, etc. This phase of the subject will be discussed in considering the nature of the various processes.

Edema.—The edema associated with nephritis is not due to a single causative factor. In the hemorrhagic types the edema may be chiefly or entirely anemic in character. With the chronic interstitial type it is of cardiac origin, and only in some of the acute forms and in chronic parenchymatous nephritis do we have actual renal edema.

The exact cause of renal edema has not been determined. The theories of water retention, chlorid retention, vascular injury, and changes in the blood, all have their supporters. The only type of experimental nephritis associated with edema is that produced by uranium nitrate, which in the main excites a tubular type of nephritis. Heinek's observation that an animal with chronic nephritis receiving serum from an animal with uranium nephritis develops edema is of extreme interest. Injury to the vessel wall by the serum, in an animal with nephritis, would appear to be the most probable explanation. In fact, recent work leads us to believe that vascular injury is a very important factor. Cohnheim and Lichtheim found that large amounts of normal salt, equal to 92 per cent. of the body weight, could be injected into an animal without producing subcutaneous edema, although ascites and dropsy of the various organs readily appeared. When the skin of such an animal was irritated by exposure to the sun's rays or by painting with iodin, typical subcutaneous edema appeared, due, according to Cohnheim, to alteration in the vessel walls by these external agents. Magnus has shown that under the same conditions of transfusion subcutaneous edema appears if the animal has previously received arsenic or a vascular poison, chloral, or chloroform narcosis. It has been shown by Schlayer that in experimental vascular nephritis the renal vessels have lost their power to dilate when excited by certain stimulants, and have diminished permeability. In all forms of experimental edema the introduction of large amounts of fluid is an essential factor. Retention of fluid, due to impaired kidney function, when combined with the increased permeability of the vessel wall, offers a very plausible explanation for renal edema. Pearce, who has studied carefully the question of renal edema, considers three factors are essential for its development—nephritis, vascular injury, and increase in the body fluids. He also determined that the edema is due to disturbed kidney function rather than reduction in the functioning area. The blood serum of patients with renal edema has a decided lymphagog action when injected into animals. The flow of lymph, according to Kast, is increased tenfold. These results have been confirmed in a measure by Starling.

The experimental work of Schlayer and Hedinger has thrown considerable light upon one of the factors responsible for edema, viz., water retention. They tested the various types of experimental nephritis to see if there is any disturbance of the vasomotor control in the kidney, determining the vasoconstrictor power of the renal vessels by electric stimulation and by adrenalin; the vasodilator reaction by the use of diuretics, as sodium chlorid and caffein. Their results indicate that in the vascular type of nephritis (that due to arsenic or cantharides) the vasomotor action of the kidney is disturbed, both constriction and dilatation being impaired; and diuresis fails to appear after the use of sodium chlorid or caffein. With that type of nephritis, where the poison acts especially upon the tubular epithelium, i. e., chromium and sublimate, little if any disturbance of the vasomotor control of the kidney has been noted. It is important to remember that in neither of these types of nephritis, when excited by the agents mentioned, is there any edema. Edema only appears after the uranium nephritis, which it is true is of the tubular type. By applying this method of investigation to uranium nephritis, they find that in those cases where edema fails to appear the kidney reacts in the same manner as the normal or as tubular nephritis due to chromium. However, in those animals which develop edema, while the reaction early and late is the same as described, there is a period, usually about thirty-six hours after the injection and corresponding to the time when edema appears, when the kidney vessels react in a peculiar manner. They still constrict and dilate when the proper stimulus is applied. Sodium chlorid and caffein still cause marked dilatation of the renal vessels, but diuresis does not follow. They have endeavored to discover the cause of the impaired permeability. Inasmuch as it does not appear in other forms of tubular nephritis, it can scarcely be due to impairment of the epithelium of the tubules. The disturbance must be, therefore, in the vessels of the glomeruli. We have here apparently an explanation of water retention in nephritis. Edema only appears in the animal experiments when the animal receives a large amount of fluid. When left on a dry diet it fails to develop.

In addition to the lessened permeability of the renal vessels and large intake of water, a third factor is apparently essential. Animals with impermeable kidneys received large quantities of water and salt and still failed to get subcutaneous edema. After waiting four to five days, then repeating the large intake of water and salt, edema developed. Evidently during this interval some change instrumental in the development of edema had taken place, and this Schlayer believes to be increased permeability of the peripheral vessels.

SUMMARY.—Summing up their results, lessened permeability of the renal vessels, increased ingestion of fluids, and increased permeability of the peripheral vessels apparently account for the subcutaneous edema in uranium nephritis.

Pearce, by a somewhat different method, has arrived at the same conclusion. He produced a tubular type of nephritis with chromate, edema, as usual, not developing. He then produced vascular injury with rattlesnake venom, ricin, or arsenic—all well-recognized vascular poisons. To produce a condition of hydremia, the rabbit received through a stomach tube 100 c. c. of water daily. By combining these three factors—renal injury, vascular injury, and increased fluid intake—he was able to cause subcutaneous edema. No one of these alone or any combination of two was sufficient. The nature of this substance, causing injury and increased permeability of the peripheral vessels in nephritis in man, is yet to be determined. In the acute scarlatinal nephritis this increased permeability may be excited either by the retained products of metabolism, the toxins of the invading microorganisms, an internal secretion of the kidney, or in the secretion from some correlated organ. Timofeew has recently explained the vascular injury as due to the products of the destroyed epithelium. This action is not peculiar to the renal epithelium, but may be produced by the dead cells of other parenchymatous organs, and he explains on this basis the interesting edema described by Cohnheim where the clinical picture is that of renal dropsy, but where the urine shows nothing abnormal and autopsy fails to show a nephritis. This peculiar edema has been observed after scarlatina, typhoid, measles, and certain gastrointestinal disturbances in children. In all of these cases more or less extensive degenerative changes have been detected in the liver or other parenchymatous organs, and he considers the products of this change responsible for the increased permeability and edema.

Widal has laid special stress on the importance of chlorid retention in the production of edema. Clinical experience has shown that restriction of salt lessens the intensity of edema. Experimental work in animals would indicate that chlorid retention does not necessarily accompany edema, and that salt is less essential than water in its production. The cause of the salt retention is still unexplained. It may be due to lessened permeability of the kidney to salt, or to primary retention of chlorid in the tissues. Castaigne's experiments apparently support the latter view. Dogs with experimental nephritis and lessened chlorid elimination were still able to eliminate chlorid in a normal manner provided they were injected directly into the renal artery. He furthermore bled normal and nephritic animals, and then injected an equal amount of normal salt solution. Shortly afterward blood was taken from the renal artery of both animals and its salt content determined. The blood from the renal artery of the nephritic dog contained less chlorid. This would indicate that in nephritis the chlorids are retained by the tissues rather than imperfectly eliminated by the kidney.

The most recent theory of edema, and one which is responsible for much discussion, is that suggested by Martin Fischer. According to Fischer, edema is due to increased affinity of the colloids of the tissues for water, this increased affinity being due to excessive acids, the result either of abnormal production or inadequate elimination. According to this theory the cause of edema is excessive acid in the tissues, the colloids present responding by swelling. The salts, including sodium chlorid, lessen the tendency of the colloids to take up water. Fischer's treatment of edema based upon this theory is to neutralize the acids in the tissues by the use of alkalies, and to administer sodium chlorid to lessen the colloids' affinity for water. For this purpose he recommends the intravenous injection of a solution containing sodium chlorid, 14 grams, crystallized sodium carbonate, 20 grams, water 1,000 c. c. The truth of Fischer's theory and the value of his therapeutic suggestions are still under discussion and it is too early to state their value.

Increased Blood Pressure.—In both acute and chronic nephritis increased arterial tension is a common occurrence. It is generally conceded that nephritis is by far the most frequent cause of the hypertension. In the parenchymatous and amyloid kidney blood pressure is not increased, but in the so-called vascular types, glomerular and interstitial forms, hypertension is the rule. This increased pressure may appear within 48 hours after the onset of an acute nephritis.

In general, a rise in blood pressure may be due to various factors—increased heart rate, increased systolic output, or shortening of the systole—so that a certain volume of blood is forced from the ventricle in less than the normal period of time. In nephritis none of these conditions is present. The viscosity of the blood may be increased so that the amount of friction against the vessel wall is greater than normal. Evidence at the present time would indicate that the viscosity of the blood in interstitial nephritis is diminished rather than increased (Bachmann, Austrian). There remains as the only plausible explanation a narrowing of the lumen of a part or all of the arterial system. On account of the rapidity with which a rise in pressure occurs, it must be due to functional narrowing. Traube maintained that constriction of the renal vessels alone was responsible for the condition. Recently Aluens has shown that compression of the kidneys causes a marked increase in the general blood pressure which continues until the pressure is removed. This interesting observation adds further support to the mechanical theory of hypertension, and also indicates how local disturbance in the kidney may cause increase in the general blood pressure. Johnson, Gull, and Sutton claim that the smaller arteries are only involved, but the weight of evidence favors constriction of the entire arterial system. One strong evidence favoring this is the frequency with which hypertrophy of both ventricles is noted, according to Hasenfeld and Hirsch in 82 per cent. of the cases. We are still in doubt as to the nature of the substance causing this increased vascular tonus. The theory has been advanced that it may be due to increased secretion on the part of the suprarenal glands. Several of the French writers have reported hypertrophy of the suprarenals in nephritis and Wiesel in addition reports increase in the chromaffin tissues in the sympathetic nervous system. With the delicate tests for detecting adrenalin in the circulating blood, an effort has been made to determine whether there is an increase in nephritis. The results have been far from uniform. Bittorf, Pal, Schlayer, Fraenkel, and others have been unable to detect any increase. Schur and Weisel, Goldzieher, and others have reported a very decided increase not only in interstitial but in the parenchymatous type. With such conflicting results it is difficult to draw conclusions, but a careful review of the work leaves the impression that those obtaining negative results have the better of the argument. A source of error is the failure to use a sufficient number of controls and thus determine the normal variations. Kretschmer has reported an increase in acute and subacute hypertonic nephritis, but a normal or subnormal amount in the chronic hypertonic forms.

Loeb explains hypertension by assuming glomerular involvement in all cases, this interfering with the proper blood supply of the kidney. The afferent vessel to the glomerulus becomes distended, this starting a reflex, the object of which is local vasodilatation. To accomplish this end vasoconstriction of the splanchnics takes place, increasing the general blood pressure. Experimentally it has been shown that marked reduction of kidney substance by successive operations leads to a permanent condition of high blood pressure. Whether this is merely the result of reduction of kidney substance, or from the nephritis excited by the procedure, has not been determined. Uremia is frequently, but not always, associated with high blood pressure, and during the acute uremic attack the blood pressure is often higher than in the free interval. Of extreme interest is the marked variation in pressure in untreated cases of hypertension. Such transitory changes would indicate that a functional disturbance is responsible for the high pressure.

SUMMARY.—Briefly summarizing our present knowledge of hypertension in nephritis, it can be said: That its exact cause has not been determined. Several theories have been advanced, none of which are free from objection. It is interesting from a therapeutic standpoint that these theories assume that hypertension is a compensatory measure; an effort to force more blood through the kidney in order to increase elimination. If this is true, the attempt to reduce blood pressure may not be good therapeutics.

Heart Hypertrophy.—Heart hypertrophy is common in both the acute and chronic forms of nephritis when associated with hypertension. An increase in blood pressure of four weeks' duration is sufficient to produce heart hypertrophy. In the majority of cases (82 per cent.) Hasenfeld and Hirsch found both sides of the heart hypertrophied. Whether we are to infer from this that the hypertension prevails in both systemic and pulmonary circulation, or whether, as maintained by Pässler, hypertrophy of the right ventricle is due to insufficiency of the left heart with consequent hypertrophy of the right, it is generally conceded that only those cases of nephritis with hypertension lead to heart hypertrophy. It is probable that cardiac hypertrophy, when due to nephritis, is always the result of increased blood pressure. Jores, who has made a study of the frequency of heart hypertrophy in interstitial nephritis, reports that in the primary contracted kidney heart hypertrophy is rarely missed, while in the secondarily contracted kidney it is commonly absent. This is in accord with clinical experience, as, in parenchymatous nephritis, which later passes into a secondarily contracted kidney, the blood pressure frequently remains normal.

Uremia.—The symptoms of acute uremia can best be accounted for on the basis of an intoxication. The clinical symptoms of nausea, vomiting, diarrhea, delirium, epileptiform convulsions, and coma can scarcely be explained upon any other basis. On account of the wide variability of the clinical picture the question has been raised by Arcoli whether the symptoms can be explained by the action of a constant or single intoxicating agent. Regarding the character of this hypothetical poison or poisons we are still in doubt. It is generally assumed that the retention in the blood of products normally eliminated by the kidney is responsible for the disturbance. The evidence to support this view is, however, far from convincing. If this were true, we would expect, after removal of the kidneys, a symptom-complex resembling uremia. In animals such removal results fatally within five or six days, yet without the symptoms of uremia. In complete anuria from obstruction to the ureter, which may persist for ten to twelve days, or even longer, before death ensues, uremia is not observed. It might be assumed that the intoxicating agent in uremia is found in such small amount that in obstructive anuria, or after removal of the kidneys, life is not sufficiently prolonged to permit of the formation of a sufficient amount to produce symptoms. Against this view is the rapid appearance of uremia in certain cases of acute nephritis. Attempts to produce uremia by introduction into the organism of large amounts of urea or sodium chlorid have been unsuccessful. It is true that in nephritis the urea is not eliminated in the normal manner; especially in uremia, the increase of urea in the blood over the normal may be very marked. If the uremic symptoms were due to this excessive urea retention, we should be able to produce them experimentally by the introduction of large amounts of urea; but such efforts have always failed, as urea so injected is almost devoid of toxic properties. Frerich, years ago, ascribed uremia, not to urea, but to ammonium carbonate formed from it by the action of a ferment. The absence of increased ammonia in the blood in uremia disposes of this theory. Creatinin and the other nitrogenous products of metabolism have been held responsible, but no one has been able to obtain any direct evidence that they play a rôle.

In mild types of nephritis it has been shown that nitrogen elimination is delayed; that is, the interval elapsing between the ingestion and excretion is considerably longer than in the normal kidney. These mild cases show comparatively little increase in nitrogen in the blood. With severe types of nephritis there is not merely delay, but absolute retention of nitrogen, so that the amount in the circulating blood is greatly increased. The more nitrogen retention the greater is the danger of uremia. In fact, it is generally considered that uremia only develops in those cases of nephritis where nitrogen retention is marked. This does not prove, however, that nitrogen retention is responsible for the uremia. Von Noorden reports a rare instance of uremia where nitrogen retention was absent. If this is true, certainly uremia may develop independent of disturbance in elimination of nitrogen. However, it has been noted that these are all patients with edema, and this may account for the apparent absence of increased nitrogen in the blood. The amount in the blood plus that in the dropsical fluid probably makes a total much greater than normal. Clinicians have recognized that uremia may develop, the result of rapid absorption of a nephritic edema associated with nitrogen retention. Müller, in answer to Arcoli's statement that complete obstructive anuria does not usually lead to uremia, lays special stress on the absence of typical uremic symptoms in those dying from acute nephritis, where conditions resembling somewhat that of the acute anuria are present. It is in the chronic nephritis that typical uremia is most frequently observed.

When we study the elimination in nephritis of the various other substances normally present in the urine, we have still less evidence that they play a rôle in uremia.

Uric acid is eliminated in nephritis more completely, as a rule, than the urea. However, the results in this respect are not uniform. The same holds true of creatin.

Of the inorganic salts, the sulphates and phosphates behave in much the same manner as the nitrogenous products. The chlorids behave in a different manner. In all patients without edema, the elimination of chlorids is satisfactory, although there may be marked nitrogen retention and threatening uremia. All nephritics with edema, on the other hand, show chlorid retention. This is not peculiar, however, to the edema from nephritis, as the same thing occurs in cardiac edema, ascites due to liver cirrhosis, pleural effusions, and in croupous pneumonia. Retention of fluid in the body is always associated with the retention of chlorids, the water retention being probably the primary. On account of the toxicity of potassium salts, Feltz and Rourke advanced the theory that they were responsible for uremia. They furnished considerable experimental evidence, but later investigators have failed to confirm their results and the theory has been abandoned.

Although it has not been proven that imperfect renal elimination is responsible for uremia, the retention theory has still the largest number of followers. Ascoli, however, believes we must direct our investigation into other channels, and has advanced the theory of nephrolysins. It has been shown that an extract of kidney, when injected intravenously into an animal, produces an albuminuria and the serum of this animal, when injected into a second animal, will excite an albuminuria. He believes in nephritis the destruction of kidney cells furnishes the material for nephrolysins, which, in turn, are responsible for the uremia. A vicious circle is established by these nephrolysins, in turn leading to further kidney degeneration; so the process becomes chronic. Ascoli and others who have repeated his experiments have never been able to produce a uremia, so the essential part of the theory is still unproven. There is no evidence that the nephrolysins are especially toxic for the nervous system, and, finally, uremia occurs in chronic interstitial nephritis, where little, if any, degeneration of kidney tissue is taking place.

SUMMARY.—Summarizing our present knowledge of uremia, the nature of the toxic agent has not yet been determined. It probably arises from impaired elimination through the kidney, and there is a strong belief, unsupported by positive evidence, that the toxic substance is some product of nitrogenous metabolism.

Value of Functional Kidney Tests.—The ability of the diseased kidney to eliminate salts and urea has been considered. It has been shown that in nephritis both salts and urea may be eliminated in a normal manner. As a rule, however, nitrogen elimination is delayed and irregular and not dependent closely upon the amount of proteid ingested. Urea determination, without a knowledge of nitrogenous intake, is valueless and misleading. Even when the proteid intake has been accurately determined the nitrogen output in nephritis may differ so little from the normal that it has little value as a functioning test. Albumin in the urine does not signify nephritis, as it may be present in considerable amount without coexisting anatomical changes in the kidney, as in the various so-called physiological albuminurias, and associated with passive congestion of the kidneys. Palpation of the kidney usually excites a transitory albuminuria. Small amounts of albumin can be very frequently detected in the urine of apparently healthy individuals. Spiegler's reagent, which detects albumin in 1-350,000, gives a positive test with practically every urine. With less delicate tests, as, for instance, acetic acid and potassium ferrocyanid, albumin is detected in only about 4 per cent. of healthy individuals. The significance of albumin as an evidence of nephritis depends partly upon the amount present, although as much as 2 per cent. has been reported by von Noorden in physiological albuminuria. On the other hand, in chronic interstitial nephritis, the albumin may be entirely absent with the ordinary tests, and is never present in large amounts in uncomplicated cases. It is, therefore, impossible, by testing for albumin alone, to determine the presence of organic changes in the kidney.

The above holds true largely in regard to urinary casts. From the time casts were discovered by Henle in 1841, until the appearance of the centrifuge in 1891, their presence in the urine was considered as positive evidence of nephritis. Since using the centrifuge, it has been determined that casts are commonly present in normal individuals. Gentzen examined a single specimen from one hundred apparently healthy individuals, and found granular casts in thirty-six, and hyalin or granular casts in sixty. Klieneberger, examining twenty-four-hour collections of urine, found casts in 78.5 per cent. These findings have been verified by many other investigators so their correctness cannot be questioned.

Exercise increases greatly the frequency with which albumin and casts appear in the urine. McFarland found abundant albumin and casts in six out of seven bicycle riders. After very severe physical labor, as, for instance, after a boat race, albumin, granular and blood casts are commonly present in the urine. For this reason albumin and casts are of much more diagnostic significance when found in the urine collected while the patient is at rest. In doubtful cases the presence of albumin and casts in the urine during a period of complete rest speaks very strongly for nephritis. It is not to be inferred that the albumin and casts are of little diagnostic significance. Especially the presence of granular casts in considerable numbers almost invariably means nephritis.

Various other tests for determining the functioning power of the kidneys have had a more or less transitory existence. Those still remaining and considered of some value are the indigo carmin, phloridzin, phenolsulphonephthalein, and tests for molecular concentration of the urine. Koranzi first used the freezing point of the urine as an index of its molecular concentration. The freezing point of distilled water is zero. As we dissolve a solid substance in it, which increases its molecular concentration, the freezing point of the solution falls. By this means we are able to determine the exact amount of solids dissolved in the urine, thus representing the ability of the kidney to eliminate these substances. While this test in many respects corresponds to the specific gravity, it is much more accurate, as two urines of the same weight may show marked difference in their freezing point. The test, however, has proven to be of comparatively little practical value, as the freezing point of normal urine varies greatly, depending upon the character of food and certain vasomotor influences over which we have no control. By determining, at the same time, the freezing point of the blood, it was thought the value of the test might be enhanced. If the kidney eliminated poorly, the retained product would modify the molecular concentration of the blood, and, therefore, its freezing point. It was found, however, that the freezing point might be lowered from other causes than impaired kidney function, as in pulmonary conditions and with cyanosis from any cause.

Another functional test which was developed, chiefly as the result of studies with molecular concentration, is known as experimental polyuria. It is based upon the ability of the normal kidney to excrete either a very dilute or very concentrated urine. This power is often impaired when the kidney is diseased. The chief value of the test, however, is to determine the latent reserve power of the kidney.

Völcker and Joseph developed an indigo carmin test, 1 1/2 to 2 2/3 grains (0.08 to 0.16 gm.) are injected subcutaneously. Within six to ten minutes the blue coloring matter appears in the urine. It has been found that in parenchymatous nephritis it is eliminated the same as the normal. The excretion is delayed and prolonged in interstitial nephritis. The chief value of the test is in comparing the functioning of the two kidneys, as by ureteral catheterization the comparative readiness with which the indigo appears can readily be determined.

The functional test of considerable value at present is the elimination of sugar after the injection of phloridzin. Casper and Richter developed this test in its application to surgical diseases of the kidney. By combining it with ureteral catheterization they were able to determine the amount of sugar excreted by each kidney. Later it was modified by Kapsammer, who laid special stress on the promptness with which sugar appeared after the injection, normally 10 to 15 minutes. It has been shown, however, that the results in the same individual vary greatly on different days. It is true that in the majority of nephritics there is considerable delay in the appearance of the sugar. The test, while probably one of the most satisfactory developed up to the present, is far from reliable.

Rowntree and Geraghty have apparently overcome many of the objections previously made in regard to functional kidney tests by the introduction of phenolsulphonephthalein for this purpose. This test originated as a result of the researches of Abel and Rountree upon the action of the phthalein family. A solution is carefully prepared containing 6 mg. of phenolsulphonephthalein to each c. c., and one c. c. of this solution is injected subcutaneously. The bladder is emptied, the catheter left in place, the escaping urine drained into a test tube in which has been placed a drop of 25 per cent. NaOH solution, and the first pinkish tinge in color of the urine noted. In normal individuals this pinkish color appears in five to eleven minutes. In addition, the total amount eliminated is determined by colorimetric methods, the color of the urine being compared with a standard solution. The solutions and colorimeter can now be purchased ready for use.

There are numerous reasons why functional kidney tests should be unreliable. In the secretion of urine several distinct processes take part: as filtration, osmosis, selective cellular activity, and absorption. These are carried on by different portions of the kidney. In the different types of nephritis the disturbance of function varies, as in the glomerular form the filtration process is interfered with, although the function of the tubules may remain normal. Again, in nephritis in man, compensating hypertrophy of the normal tissues occurs, so that from a functioning standpoint the kidney may be normal, although anatomically it may be distinctly pathological. The hypertension and heart hypertrophy may be looked upon as a compensating process, tending to bring the kidney elimination up to normal.

PRINCIPLES OF TREATMENT OF NEPHRITIS

The treatment of nephritis may be considered under the head of prophylaxis, general principles, symptoms, and complications. The general principles include, first, to avoid as far as possible everything that might tend to further injure the kidney; second, as the kidney function is impaired, to relieve it of excessive work. This to be accomplished by proper diet and by calling upon the skin and bowel to assist in the elimination of water and solids; third, the use of such measures as would tend to restore the kidney to its normal physiological function.

The treatment of nephritis is chiefly symptomatic. There are no specific remedies capable of modifying the degenerative processes in the kidney. Our efforts in the acute forms must be devoted toward relieving the kidney of its work and removing, if possible, the cause of the trouble, thus tiding matters over until the disease subsides and the physiological function of the kidney is wholly or partially restored. This restoration of function may be brought about in several ways. The acute process may subside and regeneration of destroyed tissues takes place, or the normal kidney tissue undergoes hyperplasia, or the parenchymatous nephritis passes into a secondary contracted kidney, with increased urinary output of both water and solids—a restoration in part of the disturbed function. The hypertension and heart hypertrophy developing in interstitial nephritis lead to increased elimination through the kidney, and may be considered a compensatory process. Such a patient may live for many years without any evidence of acute renal intoxication, the symptoms complained of being entirely the result of the hypertension. So complete is the restoration of function in interstitial nephritis that death is rather infrequently due to uremia, but to ruptured arteries or incompetent heart, the result of prolonged high blood pressure. In these cases treatment must be directed toward increasing elimination through other channels, thus lowering blood pressure and so regulating the patient's manner of living as to avoid overtaxing an already overworked heart.

The treatment of a chronic interstitial nephritis is chiefly directed toward the heart. In chronic parenchymatous nephritis the heart is not usually involved and the treatment is confined to the kidney. An important part of the treatment is to avoid all those things, either in the form of food, chemicals, or habits of the patient, that might irritate the kidney; to arrange a diet with the minimum amount of proteid consistent with the demand of the body, and to supply carbohydrates and fats in sufficient quantity to maintain the proper nourishment of the individual, a point quite as essential as the restriction of proteids. To facilitate elimination through the skin a warm climate and suitable clothing are important. The bowels should be kept free to assist in the elimination of water, salts, and nitrogenous waste. As the individuals are usually anemic and undernourished, too free catharsis must be avoided. For the anemia induced by the continued loss of albumin, iron or foods containing iron should be given. To lessen the edema restriction of fluids and salt is important. All of these measures are also indicated in the treatment of acute nephritis. With the kidney acutely involved, extreme precautions must be taken to prevent further injury.

Prophylaxis.—The chief etiological factors in nephritis are the acute and chronic infections, chemical poisons, either exogenous or endogenous, cold, and pregnancy. Certain of the acute infections are specially liable to induce nephritis, as scarlatina, and the utmost care should be exercised by the parents to protect their children from contracting it. The importance of exposure to cold and diet, in determining the development of scarlatinal nephritis, has been probably overestimated. The bacterial toxins are, no doubt, the exciting cause, although cold may act as an important contributing factor. Tonsillar infections are another important source of acute nephritis, and the child subjected to repeated attacks of tonsillitis should have the tonsils carefully removed. Such a procedure is equally important in the subjects with chronic nephritis, as such tonsillar infections may be important factors in the frequently observed acute exacerbations. Removal of the tonsils also lessens the danger of contracting diphtheria, which next to scarlatina is the acute infection most liable to be complicated by nephritis.

There has been considerable difference of opinion in regard to the rôle of syphilis in acute nephritis. It may be extremely difficult to determine in a given instance whether mercury or the syphilis is responsible for the trouble. However, we have authentic cases where syphilis was the etiological factor. At least, the prompt disappearance under treatment would lead to that inference.

There is a long list of chemical agents, many of which are frequently prescribed, which are capable of irritating the kidney and setting up an acute nephritis. It is important in the treatment of nephritis that drugs of this sort be avoided. It is also advisable in those acute infections liable to kidney complication that these agents be used with care. Attention has already been called to those substances used to excite acute nephritis in animals—cantharidin, chromium, bichlorid of mercury, and arsenic. In addition a number of drugs are extremely irritating to the kidney and capable of exciting acute degenerative changes in the kidney in man. The more important are potassium chlorate, salol, carbolic acid, turpentine, salicylates, lead, phosphorus, alcohol, chloroform, and ether. Even sodium chlorid and theocin in large amount may excite nephritis in some of the lower animals. Especial importance is attached to the rôle played by mercury in nephritis. The readiness with which grave kidney changes may be produced in the lower animals by the use of bichlorid impresses us with the importance of carefully watching the urine when mercury is used and the danger attending its use in patients with nephritis. Klieneberger has made a careful study of the urine in both syphilitics and nonsyphilitics during inunction cures, and calls attention to the frequent appearance within four to six days of numerous hyalin casts and their persistence for several weeks after the treatment is discontinued. Hyalin casts are not usually considered of great significance, but their frequent appearance and occasional association with red blood corpuscles and very rarely with albumin, in Klieneberger's cases, are certainly highly suggestive.

Although arsenic will produce a nephritis in animals, man tolerates it very well. The new arsenic preparation, salvarsan, from present reports, is apparently only mildly irritating to the kidney, as only occasionally have albumin or casts been reported after its use.

Both experimentally and chemically the salicyl compounds have been shown to irritate the kidney with appearance of albumin and casts (Lüthje, Klieneberger, and Oxenius). Klieneberger reports that the administration of alkalies will prevent the kidney irritation of the salicyl compounds. Salol is still more irritating and numerous cases of nephritis were observed as a result of the Woodbridge treatment of typhoid, in which salol played an important rôle. Where the surface of the skin is broken, the local application of remedies as iodoform, mercury, turpentine, pyrogallic acid, balsam of Peru, and tar preparations should be undertaken with care on account of the readiness with which they may set up an acute nephritis. The application to large surfaces of the body of turpentine, kerosene, or pyrogallic acid may cause an acute nephritis. Ether and chloroform narcosis may injure the kidney. The danger can be largely eliminated by skill in administering the anesthetic. The danger of nephritis from anesthesia is always greater when the kidney is irritated by some existing acute infection, as, for instance, operations for some of the complications of scarlatina. Here the anesthesia should be as brief as possible and the minimum amount of anesthetic used. There is no reliable evidence that large meat eaters are especially prone to contract nephritis. The same is true of those eating freely of irritating or highly seasoned foods.

Endogenous toxins arising in the intestinal canal, the result of food decomposition or bacterial products, have been considered by some as important factors. Here again, however, we lack definite evidence.

Prolonged exposure to cold and, especially, wet cold, should be avoided. Siegel has shown that dogs allowed to stand for ten minutes in ice water and then placed in a cage without drying developed typical acute parenchymatous nephritis. The urine contained albumin, casts, blood and renal epithelium, and anatomically marked degenerative changes in the kidney could be demonstrated. When the dog was placed in the cold water and then thoroughly dried before placing in the cage nephritis did not develop. The manner of action is not clear. It has been assumed by Wertheimer that the external cold constricted the renal vessels, this ischemia leading to degenerative changes.

We have too little knowledge of the agencies responsible for the kidney of pregnancy to discuss intelligently prophylactic measures. To refrain from using drugs that may act as a renal irritant and avoid exposure to wet cold is advisable. Although comparatively little is known of the importance of diet in the toxemia of pregnancy, it is advisable in threatened cases to reduce the proteids to the minimum consistent with maintaining the proper nutrition.

Emerson believes passive congestion of the kidney is an important factor in nephritis, if so during the course of acute infection, cardiovascular tonics are indicated when the blood pressure becomes low.

The appearance of albumin and casts after severe athletic exercise, as racing, raises the question whether severe exertion might set up a nephritis. It is doubtful whether such exercise, even when frequently repeated, can cause nephritis. However, in those people with hereditary tendencies such severe exertions should be avoided, as should also occupation where there is exposure to lead, mercury, and turpentine, or to the extreme changes in temperature, as, for instance, in cooling rooms. The patient with chronic parenchymatous nephritis should avoid very cold baths, as they may be a factor in exciting acute exacerbations.

Climate.—The patient suffering from acute nephritis is better off at home, provided he can have proper attention. The danger of overexertion or exposure to cold renders traveling undesirable. Change of climate is usually only to be considered in the subacute or chronic forms. The chief object of such a change is to place the patient where a warm and dry climate dilates the peripheral vessels and increases the elimination through the skin. In such a climate the patient is enabled to be out of doors, in the sunshine and fresh air, factors which perhaps have only a psychological effect, yet nevertheless are of considerable importance. The patient with parenchymatous nephritis is often free from uremic symptoms in temperate climates during the summer months. With the approach of cold weather he often becomes distinctly more uncomfortable. The effect of cold is still more marked in patients with interstitial nephritis and high blood pressure. During the cold days of late autumn they notice increased poststernal distress on walking, or this may be increased to the point of actual pain in the form of angina pectoris. They are more liable to attacks of nocturnal pulmonary edema, especially if they dress or undress in a cold room. The poststernal distress is probably due to the increase in blood pressure on a heart already overtaxed by the constriction of the peripheral blood vessels from the cold.

The chief benefit of change of climate for the patient with parenchymatous nephritis is to increase the elimination through the skin. The daily loss of water through this channel may be much greater in a dry warm climate than we are able to induce by artificial sweats. Loew has shown that the daily loss of water through the skin and lungs of a resting patient, in a dry climate like southern California, may reach 2-2½ liters (2-2½ quarts). It is true that the amount of solids in the perspiration is not very great: about 1 gram (15 grains) of urea and about the same amount of sodium chlorid per liter. However, it is quite certain that under these conditions the chronic uremia symptoms become less marked. When possible it is advisable to select a climate where the nights are not too cool. This is, however, difficult, and may be easily overcome by suitable clothing and having the patient remain indoors after sunset. Altitudes above 3,000 feet should be avoided, especially by those patients with weak hearts or high blood pressure. Patients with hypertension, who are able to walk about, should avoid hilly regions, as this may materially interfere with their out-of-door exercise. If the place selected is on the seashore, sea bathing should be avoided, as the cold and exertion are both undesirable. In this country a suitable winter climate may be found in southern Texas, southern California, the low altitudes in Arizona, New Mexico, and Mexico. The climate of the Gulf States is less desirable on account of the greater amount of moisture. Abroad, Egypt, Sicily, southern Italy, as Capri, Sorrento, Cannes, Mentone, and San Remo. Abroad, a suitable summer climate at moderate altitudes can be found in the Black Forest. In this country during the summer, the patient does best in a climate where the nights are comfortably cool as in northern Michigan or Wisconsin, and along the northern seashore. It has usually been considered that high altitudes are dangerous to patients with high blood pressure. Recently Staubli has made some observations on the effect on hypertension in patients at St. Moritz, Switzerland. He reports that continued residence at such an altitude is followed by a rather decided fall in blood pressure with gradual return to the previous level after the patient goes back to a low altitude. The patient during his residence in the high altitudes feels better, getting relief from certain cardiac disturbances, as angina pectoris, poststernal distress, and palpitation. The question arises how much of the improvement is due to high altitude per se, and how much to mental relaxation and psychical effect of a visit to a place like St. Moritz. Staubli's observation, however, if verified, shows that the dangers of high altitude for this class of patients may have been overestimated.

Climate does not cure the chronic forms of nephritis, but it makes the patient much more comfortable and not infrequently prolongs his life by preventing acute exacerbations and lessening the tendency to uremia.

Rest.—The patient with acute nephritis should have absolute rest in bed. This insures uniform warmth to the surface of the body, limits body nitrogenous waste to the minimum and reduces the work of the heart. Standing or exercise increases the amount of albumin and number of casts. The increased nitrogenous waste with moderate exercise is almost imperceptible, and is of little importance. The rather decided rise in blood pressure frequently observed very early in the glomerular type of the acute nephritis increases very materially the work of the heart. Rest until the necessary hypertrophy has taken place is very important. Mental strain, as it raises the blood pressure, should also be avoided.

The patient with chronic parenchymatous nephritis should observe quite closely the same precautions in regard to rest as the individual with the acute form. When edema is present little exertion should be indulged in. To obtain fresh air, sitting out of doors or riding, in suitable weather, may be permitted. At all times protection from the cold must be observed. To carry out such precautions for months or even years is often difficult, but it is important if we wish to avoid the acute exacerbations so commonly seen in this type of nephritis. The benefit derived from prolonged rest in bed is frequently observed in patients in a free public hospital, where they may remain in bed or at least indoors and quiet for months or even a year or more, with gradual improvement. As a rule, when such a patient leaves the hospital he will return within a few weeks with all the old symptoms and another prolonged stay in the hospital is necessary. Rarely does a patient kept quiet in this manner suffer from the acute exacerbations so commonly observed in the parenchymatous type of nephritis. In the chronic interstitial nephritis the same rules, as regards rest, should be observed as are commonly employed in weak hearts. On account of the increased work thrown upon the heart by the high blood pressure, overtaxation should be carefully avoided. Just the degree of rest is dependent upon the working power of the heart. Where incompensation is marked, absolute rest in bed may be necessary. In other cases it is only advisable to avoid hill or stair climbing, or heavy lifting. A safe guide for the patient is the appearance of dyspnea, and he should be taught to avoid all those things which cause shortness of breath. It is much easier to prevent overtaxation and dilatation of the heart than it is to restore it again to its previous condition. When cardiac insufficiency develops, the result of hypertension, the probability of lasting improvement is not very great. In many of these cases the high blood pressure can be very slightly or not at all modified, and it is very difficult to restore a heart which is constantly compelled to do increased work. Massage may be used advantageously in the chronic form of nephritis. Where the heart is weak it should be given lightly, and when it excites fatigue or cardiac disturbance should be discontinued or given less vigorously. The use of fats, as cocoa butter, commonly employed, should be avoided, as they interfere with proper elimination through the skin.

Bathing and Care of the Skin.—Proper functioning of the skin is very important in all forms of nephritis. To maintain this, frequent bathing is essential. In nephritis prolonged cold baths are contraindicated. In acute and parenchymatous forms, because cold temporarily constricts the peripheral vessels, causing hyperemia of the splanchnic circulation, including the kidney, and by interfering with the secretion from the skin. With interstitial nephritis the cold contracts the skin vessels and raises blood pressure. When given, except for the purpose of sweating, baths should be as near the body temperature as possible. There are no special advantages to be derived from medicated baths.

Elimination Through the Bowel.—Good elimination through the bowel is essential in all cases of nephritis. With impending uremia or edema very free catharsis is important. By free catharsis the amount of nitrogen eliminated through the bowel may be greatly increased. While normally ten per cent. or less of the nitrogen ingested leaves the body in the stool, in uremia patients with diarrhea this may reach thirty per cent. (v. Noorden, Ritter). This vicarious action of the bowel becomes, therefore, of considerable importance. The normal daily feces contain less than one grain of sodium chlorid. By means of free catharsis this may be increased to 4 or 5 grains daily, a matter of considerable importance in those cases where the kidney is impermeable to salt. The chief value of free catharsis, however, is to get rid of fluid, and for this reason it is especially indicated where edema is present. Judgment must be exercised when employing this means to dispel dropsy. The edematous patient may be quickly freed of his dropsy by excessive catharsis, but left in such an exhausted state that his condition is more serious than before. Especially prolonged catharsis may do harm by impairing nutrition. By proper restriction of fluids and salt, and by placing the patient on a non-nitrogenous diet, extreme catharsis is rarely required. The cathartic selected should produce a watery stool, and at the same time not irritate the kidney. On account of their irritating action, mercury preparations should be avoided. A saline cathartic is quite as efficacious as calomel, and safer. In selecting a saline cathartic palatability should be considered. Sodium phosphate in doses of 4 to 20 gm. (1-5 ʒ) dissolved in hot water is reasonably palatable. Calcined magnesia given either in the powder form in 1 to 4 gms. (15-60 grs.) or in the form of milk of magnesia, 15-50 c. c. (½-2 ʒ) is usually well borne. The liquor magnesia citrate (U. S. P.), while reasonably palatable, is somewhat objectionable on account of fluid contained in dose, 360 c. c. (12 ℥). The sodium and magnesium sulphates, while efficient, are extremely distasteful to most patients. Some patients prefer some of the bottled saline waters, as Apenta or Hunyadi. Saline waters containing large amounts of sodium chlorid, such as Pluto water, should be avoided where there is evidence of salt retention. The effervescing salines, as effervescing phosphates, or Seidlitz powders, while palatable, are objectionable on account of the amount of gastric distention. This distention is especially dangerous in dropsical patients with ascites, where the stomach is compressed below, and the enlargement forces the diaphragm upward. The salines are best taken in the early morning on an empty stomach, and preferably in half a glass of hot water. The milk of magnesia, on account of its palatability, may be taken at frequent intervals during the day.

Of the vegetable cathartics, jalap and elaterium are most frequently recommended on account of producing a watery stool. Jalap is usually given in the form of compound jalap powder, pulvis jalapæ compositus (U. S. P.), in dose of 1-4 gm. (15-60 gr.). Elaterium may be given in pill form in dose of 10 mg. (1-6 gr.), or as elaterin, 2-4 mg. (1-30-1-15 gr.). Both jalap and elaterin are more apt than the salines to cause griping. Any of the other vegetable cathartics, as cascara sagrada, rhubarb, aloes, or senna, may be used, but are less desirable, as they do not produce a watery stool.

Recently phenolphthalein, dose 0.1-0.2 gm. (1½-3 gr.), has been advocated as a laxative, but the action is like cascara or rhubarb.

Restriction of Fluids in Nephritis.—Until comparatively recently it has been the custom to give patients with nephritis large amounts of fluid. In the acute and chronic parenchymatous types it was thought by this means to flush out the kidney and free the secreting tubules of casts, epithelial cells, and other débris. Toxins were by this means eliminated in a more dilute form and rendered less harmful to the kidney. In the chronic interstitial nephritis, the kidney having lost its power to excrete a concentrated urine by increasing the output of water, there would be a correspondingly increased elimination of solids. It has been shown, chiefly through the work of von Noorden and his associates, that in certain forms of nephritis, water drinking may be harmful rather than beneficial. With the dissemination of von Noorden's views, many have concluded that restriction of fluids is indicated in every case of nephritis. It is necessary, however, to discriminate, not merely in regard to the class of cases where such restriction is indicated, but also the amount of fluids allowable.

In every case of nephritis where the ability of the kidney to eliminate water is seriously impaired, as shown by the presence of renal edema, restriction of fluids is indicated. The ingestion of large amounts of fluids under these circumstances only increases the edema. In some patients, where edema is absent or at most very slight, the ability of the kidney to eliminate water is limited. Such a patient, if given a large amount of fluid, will develop an edema. When in doubt regarding the permeability of the kidney to water, measure carefully the fluid intake and compare it with the output through kidney and bowel, allowing 500 c. c. for the loss through the skin and lungs, or, what is more satisfactory, weigh the patient. Any decided gain in weight when on a restricted diet probably means retention of water in the tissues. In the parenchymatous type of nephritis the urine, even after drinking large amounts of fluids, remains rather scant in amount and of high specific gravity. The permeability of the kidney to water is so impaired that only a concentrated urine can be excreted. As a rule, in those cases, drinking large amounts of fluids does not increase materially the amount of solids eliminated, the water remaining in the tissues.

The daily excretion of urine in chronic interstitial nephritis is in excess of the normal, and when edema appears, it is usually of cardiac origin. In this type of nephritis the kidney has lost its ability to excrete a concentrated urine. Its permeability to water, however, is normal. It is important in these cases to give a sufficient amount of fluid to carry off the requisite urinary solids. It is desirable to determine approximately the amount of fluids required, rather than attempt to flush out the kidney by large amounts, and thus still further tax an overworked heart. Mohr has worked out this phase of the subject, and his results furnish us with a safe guide. Dennig and Spiegler determined that marked restriction of water was followed by nitrogen retention, if on the day following large amounts of fluids were given the previously retained nitrogen was eliminated, giving in this day an excess over the amount of nitrogen ingested. Mohr determined that in any form of nephritis practically the maximum amount of solids was eliminated if the patient passed from 1,250 to 1,500 c. c. of urine. The average patient, besides the fluid taken as such, receives from 500 to 750 c. c. of liquids in his other nourishment. The urinary output is about 70 per cent. of the total fluids ingested. A patient, therefore, drinking 1,500 c. c. of fluids, and taking an addition of 500 c. c. in his food, should pass about 1,500 c. c. of urine daily. This, of course, can be greatly reduced by free catharsis or sweating. A safe rule to follow is that when the kidney is permeable to water in the normal manner, i. e., no evidence of edema, 1,500 c. c. of water daily is sufficient. v. Noorden recommended in the chronic interstitial nephritis, when restricted to this amount of fluid, to give once a week a flushing out day, to carry off any retained waste. On this day the patient may take 3,000 c. c. of fluids. It is at times difficult to restrict patients with interstitial nephritis to 1,250-1,500 c. c. of fluids daily. With perseverance, and by taking small amounts of fluids at a time, drinking it slowly and avoiding salty foods, in the majority of cases, this limitation can be borne without hardship. Patients with severe renal edema may require much less fluid than the above mentioned amount. The requisite quantity should be determined by studying each case in order to determine the permeability of the kidney to water. If by administering water there is not a corresponding increase in the urine we are simply increasing the edema. Patients with uremia and marked edema may be given advantageously only a pint of fluid in the form of milk or cream daily, until the urinary flow is increased. Then gradually increase the fluid intake, but not exceeding 1,500 c. c. daily.

There are no advantages in special waters. Pure water has all the advantages of the more expensive medicated forms. The popular belief that patients with nephritis may be benefited by going to certain springs and drinking freely of the waters is fallacious.

SUMMARY.—The maximum amount of waste can be eliminated through the kidney, if the patient receives from 1,250 to 1,500 c. c., 1¼ to 1½ quarts, daily. Patients free from edema may have once a week a flushing out day when they receive double the above amounts. Patients with marked renal edema should take, as a rule, not more than 1,000 c. c. of fluid daily. If the urine is very scant, and edema marked, half of this amount may be given. The presence of renal edema is evidence of inability of the kidney to eliminate water. To administer large amounts of fluids simply increases the edema.

Restriction of Sodium Chlorid.—A few years ago Widal and Javal called attention to the inability of many nephritics to eliminate sodium chlorid in a normal manner. The salt retained in the tissues requires a certain amount of water to maintain it in the proper molecular concentration, thus leading to edema. Widal considered that chlorid retention was responsible for the dropsy in nephritis. In some cases of nephritis the kidney was permeable to urea, phosphates, and sulphates, but not to chlorids. For this reason he considered the chlorid elimination a better index of the functioning power of the kidney than the excretion of urea. Although Widal probably overestimated the importance of chlorids in edema, his teachings have been very valuable, as there is no question that in many cases edema may be reduced by the restriction of sodium chlorid. It is only necessary to weigh a patient with renal edema, who has been partaking freely of salt, then place him on a salt-free diet and notice the rapid loss in weight. Not every patient, however, with parenchymatous nephritis shows chlorid retention, and many normal individuals have very marked inability to excrete chlorid, and after the ingestion of considerable quantities of salt show a marked gain in weight. This retention may be quite equal to that observed in cases of mild nephritis. At the present time it appears that chlorid retention is only marked in severe acute or chronic parenchymatous nephritis. In the milder forms and in the interstitial type chlorid elimination approaches the normal.

Widal also considered that the chlorids had considerable influence on the degree of albuminuria and the tendency to uremia. The observations of Blookers and others have failed to confirm this. Blookers' patients did not show an increase in the albumin when sodium chlorid was given freely. The amount of chlorid consumed by the average individual is far in excess of the amount required by the body. Two, or at most three, grams is all that can be utilized, and any in excess of this amount is promptly eliminated. The amount taken daily on the average is 6 to 10 gms. A safe rule to follow in nephritis is to give the patient only the amount of salt demanded by the tissues, viz., 2 to 3 gms., simply avoiding the excess. It is rarely, if ever, advantageous to reduce the chlorids below this point, and certainly never for long periods of time. This can be accomplished by avoiding meat soups which may contain 6 to 8 gms. of sodium chlorid per liter. Their high chlorid content, the presence of extractives, large amount of water and small amount of nourishment render them especially undesirable. Salt should not be added to the food, except to butter and the small amount present in the bread. If the patient does not object to unsalted butter, this may be used. If meats are permitted, they should be fresh, not salted. If the patient's appetite is poor, to render food unpalatable by withholding salt may do more harm than good. There is, on the other hand, little danger of the patient receiving less than the physiological requirement, as one liter of milk contains almost two gms. of salt, which gives the patient all he needs. The following table shows the salt content of some of the commoner articles of food. This refers to the amount of salt naturally present. These figures are taken from Strauss' book on dietetics:

	Percentage of NaCl		Percentage of NaCl
Milk	0.16 Gms.	Blueberries	0.02 Gms.
Butter, salted	1.00	Apples	0.06
Butter, unsalted	0.02	Oranges	0.04
Cheddar cheese	1.01	Oatmeal	0.16
Edam cheese	2.57	Rice	0.10
Cream cheese	2.00	Buckwheat	0.06
Holland cheese	2.60	Nestlé's food	0.29
Eggs	0.14	Barley	0.40
Caviar, Russian	6.70–7.2	Radishes	0.02
Lean beef	0.10	Celery	0.31
Oysters	0.59	Cucumbers	0.07
Fresh fish	0.10	Spinach	0.21
Ham, raw	4.10–5.8	Potatoes	0.06
Ham, cooked	1.80–5.3	Mushrooms	0.06
Corned beef	2.04	Cabbage	0.08
Mackerel, salted	11.42	Lettuce	0.13
Liebig's extract	3.49	Asparagus	0.83
Peas	0.06	Artichokes	1.27
Beans	0.05	Tomatoes	0.14
Cherries	0.01	String beans	0.83
Grapes	0.01	Plums	0.003
Gooseberries	0.05	Apricots	0.004

AMOUNT OF SALT CONTAINED IN THE AVERAGE PORTION OF FOOD FOR ONE PERSON AS USUALLY PREPARED

	Percentage of NaCl		Percentage of NaCl
Filet of beef	3.10 Gms.	Potato soup	1.38 Gms.
Roast beef	2.80	Spinach	2.90
Liver	4.10	Cabbage	0.50
Roast pork	2.30	Mushrooms	0.50
Cooked fish	4.00	Lettuce salad	0.78
Bouillon	2.60	White bread, 100 gms.	0.40–0.7
Cream soup	0.70	Rye bread, 100 gms.	0.75
Cereal soup	1.85	Zwiebach, 100 gms.	0.38

SUMMARY.—In all forms of nephritis associated with renal edema restriction of sodium chlorid to 2 or 3 grams daily is desirable. This amount gives the patient his full physiological requirement. A salt-free diet is only necessary where edema is marked, and should not be maintained for a prolonged period. Chlorid restriction is not necessary in pure chronic interstitial nephritis, as renal edema is rarely present. Sodium chlorid does not increase the albuminuria.

Sweating.—There can be no question of the value of sweats in certain forms of nephritis. It is probably true that the benefit derived is not entirely dependent upon the amount of water and solid material eliminated. Many patients with cardiac dyspnea, due to high blood pressure arising from interstitial nephritis, are greatly relieved by profuse sweats. There may be no evidence in such a patient of renal edema, and the elimination through the kidney may be normal. Here the relief obtained is due to dilatation of the peripheral vessels thus relieving the heart. This lowers blood pressure, thus relieving the left heart of its excessive work, and the dyspnea disappears. For the cardiac dyspnea often appearing in the evening or during the night, due to hypertension, there is no other measure that so frequently gives such striking relief as sweating. In these cases the sweat should be given a short time before the usual hour at which the dyspnea appears. Daily sweats often affect favorably the high blood pressure. In a number of cases where the blood pressure has been followed carefully, the author has noted more or less permanent reduction in blood pressure after daily sweating. It is true, after the sweats have been discontinued, the pressure soon returns to its previous level. During the first week or ten days after the sweats are begun, there is only a transitory fall in pressure immediately after the sweating, varying from 20 to 40 mm. of mercury. Finally, however, the pressure may remain lower during the entire interval between the sweats. In a patient where the pressure had never been lower for a period of two years than 220 mm., after taking daily sweats for twelve days, he showed a pressure of 180 twenty-four hours after the sweat. Shortly after the sweats were discontinued the pressure returned to the previous level. After the sweat, the patient should not be exposed to the cold. In the case of a patient of mine in a hospital, the attendant in the hydrotherapy room, who had been accustomed to treat chiefly nervous patients, turned on the cold spray, causing the patient to faint. The day following, the same procedure was gone through with. Taking the patient's blood pressure, previous to the sweat, it had been 220, after the sweat it was 180. When the cold spray was applied it promptly rose to 260.

Most frequently, however, sweating is used with patients with edema either to lessen or to relieve uremic symptoms. The amount of water lost in a profuse sweat may be as much as one liter (Kovesi, Strauss). Kovesi has shown that there is included 8-9 gms. of solid matter; 2.86 gms. of which was sodium chlorid and 2.08 gms. nitrogen. Strauss studied the perspiration in 63 cases, chiefly nephritis, and found the amount of sodium chlorid varied from 0.3 to 0.7 per cent. The amount of water varied from 200 to 800 c. c. With the latter amount, if it contained 0.7 per cent. sodium chlorid, the patient would eliminate in the sweat 5.6 gms. This amount is, however, far above the average, which is not more than 2 gms. Strauss found the amount of nitrogen in the sweat varied from 1-2 gms. per liter, about one-third of which was urea nitrogen.

Leube and others have expressed a fear that in withdrawing such a large amount of fluid from the body, the increased concentration of those substances exciting uremia might precipitate an attack. This fear is not supported by clinical experience. Strauss, who has had very extensive experience, has never seen, after a sweat, evidence of increased intoxication. Inasmuch as the percentage of chlorids and urea in the perspiration approximates closely that found in the circulatory blood, there can be little danger of exciting acute uremia by sweating.

Regarding the danger of shock from the marked dilatation of the peripheral vessels, there is little to be feared, especially if the patient is lying down when taking his treatment. It is advisable when the first sweat is given to have the physician present. After this, any intelligent person can safely supervise it. If the pulse become poor, or the patient complain of faintness, the heat should be discontinued and the surface of the body bathed with alcohol. The slight degree of cold thus produced is sufficient to constrict the skin vessels and restore the balance of the circulation.

Various methods may be employed for inducing the sweat. The chief consideration is to use a means which will produce the most profuse perspiration with the least inconvenience to the patient. In hospitals or institutions with hydrotherapeutic facilities an excellent method is to place the patient in one of the large ovens used for the purpose. A cold towel should be applied to the head and the sweat continued for twenty minutes. The patient may then be removed from the oven, wrapped in blankets and allowed to cool gradually, or what is more comfortable is to rub the patient off with alcohol, avoiding chilling of the body and then cover him up warmly with blankets, these to be gradually removed.

In the home, either the hot pack or hot-air bath may be used. The former does not require any special apparatus and the results are satisfactory. A dry sheet and blanket are placed over the patient, then hot-water bags or bottles or hot bricks placed along the sides of the trunk and extremities and then several blankets carefully tucked in and spread over all. A cold cloth should be applied to the head. It may be necessary to renew the hot-water bottles, but, as a rule, this is unnecessary. The average duration of the sweat is twenty to thirty minutes. Care must be taken to protect the patient from burns. The hot-air bath may be either dry air or steam. The patient in bed is covered over with blankets, which must be supported on a frame so as to allow free space for the hot air to circulate. If a steam bath is to be given, a teakettle, with an alcohol lamp, gas, or electric heater, is placed on a chair at the foot of the bed, and the steam conducted underneath the blankets. If dry hot air is used, this can be conducted underneath the bedclothes by means of a small stovepipe about three inches in diameter—two short lengths with an elbow. To the lower end of the pipe a small hood may be riveted, in which the alcohol lamp is placed. The portion of the pipe extending under the bedding should be covered with asbestos to prevent burning the patient or bedding. Here, as with the steam, a frame should be placed about the patient to support the blankets, so as to permit of free circulation of the air. A small portable cabinet may be used, but it is not as safe or comfortable for the patient as a sweat given with the body in a horizontal position. Brieger and Diesselhorst claim to have obtained a more concentrated perspiration, i. e., containing more solid waste, when they used electric lights for generating the heat. Some patients will sweat very satisfactorily if placed in a hot bath, then put to bed and covered up well with blankets. Pilocarpin, on account of its depressing effect, is rarely desirable.

Summary.—Sweats are indicated in patients with marked renal edema, threatened uremia, or where extreme hypertension exists. The amount of water and solid constituents eliminated in a sweat may be sufficient to lessen an edema or alleviate a threatened uremia. The manner in which the sweat is given is of little importance. The only points to be considered are its efficacy and the comfort of the patient.

Diet.—Diet is one of the most interesting problems in the therapy of nephritis. Formerly the strict milk diet was considered the only safe one. With increased knowledge of the functioning power of the diseased kidney, a more liberal diet is now permitted. In arranging a diet the following points should be considered:

First—To see that the patient receives a sufficient amount of food, and that it contain at least the minimum amount of the various food principles.

Second—To avoid all the foods or drinks that contain substances which, in the course of elimination, irritate the kidney.

Third—To avoid an excess of those foods the waste of which is eliminated chiefly through the kidney, viz., the proteids. In considering this point it will be necessary to determine in a general way the ability of the kidney in nephritis to eliminate nitrogen.

Fourth—To avoid, at least in certain cases, foods containing large amounts of sodium chlorid.

Metabolism proceeds in nephritis in the same manner as it does in the normal individual, and the rules governing the required amount of the various food principles are the same. There is no ground for believing that a nephritic can get along safely upon a smaller amount of albumin than the healthy person. If not given the requisite quantity, he draws upon the body albumin. It is true, for a short period of time, the amount of albumin may be restricted; and if it is determined that the ingestion of proteids leads to retention of nitrogen, then he should be restricted to the minimum amount of albumin that is necessary to protect him from using up his muscle proteid. The amount of albumin in the urine varies greatly from day to day, even on a fixed diet; and considerable care must be exercised in drawing conclusions from the degree of albuminuria. Voight's figures have been generally accepted, that the minimum daily consumption of proteid should not fall below 85 gm. Chittenden considers these figures far too high, and has demonstrated that a working man may live on 40 gms. of proteid daily. Taking 50 gms. as the minimum daily requirement, this would be contained in the following amounts of the various foods in the fresh uncooked state:

Hen eggs	7			
Milk	1,660	c. c.	1½	qts.
Lean beef	220	gms.	7⅓	oz.
Ox tongue	300	“	10	“
Lean veal	260	“	8⅔	“
Calves' liver	260	“	8⅔	“
Lean mutton	250	“	8⅓	“
Lean pork	240	“	8	“
Very fat bacon	3,000	“	100	“
Chicken's breast	250	“	8⅓	“
Pigeon	210	“	7	“
Goose	320	“	10⅔	“

Rabbit	200	gms.	6⅔	oz.
Venison	230	"	7⅔	"
Trout, pike, herring	260	"	8⅔	"
Salmon	230	"	7⅔	"
Lobster	300	"	10	"
Oysters	800	"	26	"

COOKED BOILED MEATS

Lean beef	150	gms.	5	oz.
Lean veal	190	"	6⅓	"
Lean mutton	160	"	5⅓	"
Lean pork	175	"	5½	"
Chicken breast	160	"	5⅓	"
Salmon	210	"	7	"

BAKED OR FRIED MEATS

Roast beef	200	gms.	6⅔	oz.
Beefsteak	200	"	6⅔	"
Stewed beef	160	"	5⅓	"
Roast veal	200	"	6⅔	"
Lamb chop	220	"	7⅓	"

The individual of average weight will require, if at rest, about 2,200 calories daily. If working, this will be increased to 2,800 or 3,000 calories. If the patient is confined to the minimum amount of albumin—50 gms.—he obtains from this source about 200 calories. The remaining 2,000 or more calories he takes in the form of carbohydrates and fats, and these must be taken abundantly. As the pure fats and sugar do not contain any proteid, these may be given very freely. Each gram of sugar is equal to 4 calories. This the patient may take in the form of sugar or in jellies. A patient, provided it does not excite digestive disturbances, may take daily, in one form or another, 250 gms. of sugar, equal to 1,000 calories. The fats may be given in the form of butter and cream, and if the patient is taking meats, in the form of fat meats or oil on salads. One half liter (one pint) of 20 per cent. cream is equal to 1,080 calories, while the same amount of milk is equal to only 325 calories. Cream contains slightly more albumin than milk, but on account of its high caloric value, a patient may be properly nourished with it and still receive the minimum albumin requirement. A patient on a pure milk diet, in order to obtain 2,200 calories, must drink 3.4 liters, or about 3½ quarts. In this amount he would receive 102 gms. of albumin, or more than twice the minimum requirement. If he takes 20 per cent. cream, only one liter (one quart) would be necessary to give him 2,200 calories, and this would contain less than 40 gms. of albumin. For this reason cream is much more suited for nephritis than milk. The cream may be given in the form of cream soups, with cereals or bread, or made into a palatable drink by diluting with equal parts of seltzer or Vichy.

The various cereals contain from .7 to 12 per cent. of proteid. Peas and beans, 23 to 25 per cent. The various breads from 6 to 8 per cent., puddings from 3 to 6 per cent., soups ½ to 2 per cent., with the exception of pea, bean, and cream soups, which have 3 to 4 per cent. Green vegetables contain from 27 to 35 per cent. The root vegetables rarely contain more than 2 per cent. of albumin. The fruits contain from 0.3 to 2 per cent. of albumin. Most of them less than 1 per cent. Nuts vary considerably in the amount of albumin. Almonds, 34 per cent.; chestnuts, 10.7 per cent.; walnuts, 15.7 per cent.; hazel nuts, 17.4 per cent. Cream cheese contains only 2.8 per cent. of albumin, but Stilton, Camembert, Swiss cheese, Edam, Roquefort, and Holland cheese contain from 22 to 29 per cent.

Extreme restriction of the proteid is only advisable in the acute stage of nephritis. As soon as recovery is well advanced, this amount should be increased to 85 gms., or, according to von Noorden, to 100 gms. daily. In the chronic form of nephritis it is not advisable to restrict the patient to less than 85 gms. of proteid daily. If the restriction is carried beyond this point, the body proteid is used up to supply the deficit. The problem is in reality not how much nitrogen the kidney is able to eliminate, but how much albumin must be given to protect destruction of body proteid. The ability of the kidney in cases of acute nephritis to eliminate nitrogen varies greatly. The functioning power of the kidney in this respect cannot always be determined by the intensity of the clinical symptoms. Kovesi has shown that some patients with parenchymatous nephritis are capable of eliminating 14 to 15 gms. of nitrogen daily, equivalent to 85 or 90 gms. of proteid. In other cases, clinically not more severe, not more than 35 to 40 gms. of proteid could be disposed of. Mohr has shown that a patient with severe acute nephritis can usually dispose of 50 gms. of proteid and often of a much larger amount. Apparently, a safe rule to follow with our present knowledge, is to restrict the proteid to the minimum amount required by the body. For short periods of time 50 gms. will suffice; for prolonged periods, however, this should be increased to at least 85 gms.

The Form in Which the Proteid is Taken.—The belief has been prevalent that the diet in acute nephritis should consist chiefly of milk. This practice is not based on its digestibility, but with a view of lessening the work of the kidney. As a quart of milk contains 30 gms. of proteid, in order to keep the patient properly nourished an excess of proteid must be given, and thus the kidney is overtaxed. As the proteid restriction is the important factor, is there any objection to giving it in the form of egg, meat, and vegetable albumin? This is not merely a question of theoretical interest, but of practical value, as the patient on a pure milk diet, if properly nourished, must take at least three liters, giving thus an excessive amount of proteid. In addition, some patients object to milk, and most of them soon tire of it as an exclusive diet. Considerable work has already been carried out on this phase of the subject, and the results are quite uniform. The chief difference in the form in which nitrogen is eliminated on a meat or a milk diet is that in the former more nitrogen appears in the form of nitrogenous extractives, as creatin. Whether white meats contain less of these extractives than dark meats has not been determined. Although considerable work has been done upon these subjects, the results are so conflicting that it is impossible to draw any definite conclusions. It is, however, probable that the amount of extractives in light and dark meat is practically the same, as it is only on this basis we can explain the conflicting results. Kaufman and Mohr have studied the effect of light and dark meats on the nitrogen elimination, albuminuria and clinical course of chronic parenchymatous and chronic interstitial nephritis. They conclude that it is a matter of indifference whether white or dark meat is taken. These results have been confirmed by von Noorden, Pabst, and others. The teaching that white meat is preferable to dark meat in nephritis is so thoroughly disseminated that it cannot be uprooted at once. However, increasing evidence is being collected that this view is fallacious.

Another interesting point is the effect of meats on blood pressure. Dale and Dixon and Barger and Walpole have found that decomposing meat contains a pressor-raising substance belonging to the amino group. These pressor substances may also be formed by the action of intestinal bacteria on tyrosin. It is possible changes of this character may take place in the intestines of man, and there is some clinical evidence that a person partaking freely of meat has a higher blood pressure than when on a meat-free diet.

There can be no objection to albumin in the form of eggs, as they are free from the extractives which are found in meats. To avoid alimentary albuminuria, the eggs should be cooked. As hen eggs contain about 6 gms. of albumin three or four may safely be taken daily.

Foods directly irritating to the kidney should be avoided. This is especially important in the acute forms of nephritis. Our actual knowledge of this subject is very meager, but, following custom, certain foods or condiments may be considered harmful. In this list may be included the meat extractives which are present in all meat soups; and their use in acute nephritis is harmful as, in addition to the extractives, they contain large amounts of sodium chlorid, have little nutritional value, and increase materially the fluid intake. Smoked meats are also objectionable. Condiments, as pepper, mustard, horseradish, and the various other meat sauces, are considered renal irritants. Certain vegetables, such as radishes, onions, and celery should be avoided. Von Noorden considers asparagus harmless, at least when taken in moderate amounts. There is no objection to vinegar and lemon juice.

Coffee, Tea, Tobacco.—There is no good reason why coffee and tea in moderate amounts may not be taken, even by patients with acute nephritis. The former view, that caffein caused diuresis by stimulating the renal epithelium, has been disproved. Patients with nephritis are often needlessly restricted in their diet, especially in the withdrawal of tea, coffee, and tobacco. There is no reason to believe that smoking in moderation will have any effect upon the kidney. The effect of smoking on blood pressure varies. In some individuals there is a fall; in others a moderate rise. It is well known that smoking will disturb the cardiac rhythm, especially when indulged in too freely. Coffee, tea, and tobacco are objectionable when used to excess, and to avoid this it may be advisable to entirely forbid their use.

Alcohol.—Alcohol rather rapidly passes into the tissues from the blood, and is there slowly oxidized. Traces of alcohol may appear in the blood for 24 hours after its ingestion. Small amounts of alcohol may be excreted unchanged through the kidney. There is still considerable difference of opinion in regard to the effect of alcohol on the kidney in acute nephritis; and the rôle it plays in the development of interstitial changes in the kidney. Until our knowledge of this subject is more definite, it is advisable to prohibit its use in all forms of nephritis. An exception may be made in the chronic alcoholics who develop nephritis, as this class of patients do not tolerate well the complete withdrawal of alcohol. When given it should be in a dilute form and in very moderate amounts.

Summary.—The dietary treatment of nephritis consists, first, in giving the patient the minimum amount of proteid that can be allowed without leading to destruction of body proteid.

Second: To avoid foods or drinks directly irritating to the kidney, as certain sharp vegetables, condiments, and alcohol.

Third: Furnish the patient with sufficient food to meet the body demands. For short periods of time the diet may be greatly restricted, as in uremia, with anuria, 500 c. c. (one pint) of cream daily. Although apparently it is a matter of indifference in what form the patient receives his proteid, in acute nephritis the most satisfactory form is as milk or egg albumin, as meats contain extractives which are thought to irritate the kidney. In all forms of nephritis there is no objection to the free use of fats and carbohydrates, and with the restriction of proteids these should be given freely. Sugar, jellies, cooked fruit, with plenty of sugar, have a high food value, and are free from proteid, so may be given in acute nephritis. Cream is much better than milk on account of its high caloric value, enabling a patient to receive sufficient nourishment without excessive albumin. There are many objections to the prolonged use of a pure milk diet. It furnishes too much fluid and too large an amount of albumin.

In chronic nephritis there is no objection to the use of a moderate amount, 250 grams, 8 oz., of meat daily. It is a matter of indifference whether this be light or dark meat, chicken or roast beef. The proteid in chronic nephritis should not exceed 85 grams daily. Alcohol is only permissible in those addicted to its use, and then in moderate amounts, well diluted. Coffee and tobacco, if properly restricted, probably do little harm.

Treatment of Hypertension.—The nature of the changes responsible for the high blood pressure in nephritis has already been considered. In the pure parenchymatous type of nephritis in man the blood pressure is rarely increased. It is especially in the vascular types, acute glomerular and chronic interstitial form, that hypertension prevails. The increased blood pressure may be considered an effort to pass more blood through the kidney, and thus increase elimination by this channel. Loeb's work on this subject is especially interesting, as he found that lowering the blood pressure 35 to 45 points by nitroglycerin diminished the elimination of both water and solids. If further investigation supports this view, then lowering blood pressure by drugs may be harmful rather than beneficial. However, when the pressure becomes extreme—200 mm. or more—the danger to the heart and blood vessels is so imminent that we may be justified in attempting to reduce it. Even with lower blood pressure, if the heart gives evidence of being overtaxed, measures to reduce the pressure should be instituted. In undertaking investigation on the effect of various agents in reducing hypertension, it must be borne in mind that the normal variation in pressure in this class of patients may be very great. When the pressure is taken daily for several weeks, fluctuations of 30 to 40 mm. are not uncommon. One patient studied by the author had for one week a blood pressure of 200, then without apparent cause it fell to 110, where it remained for a period of two weeks. Following this and without any change in the treatment it rose to 220, and at this time the patient suffered from marked uremic symptoms. Such extreme fluctuations are extremely rare. The chart on page 46 shows the fluctuation in pressure extending over a year in a bedridden hemiplegic, with interstitial nephritis. Some of the high readings could be accounted for by worries, as the patient had family troubles which at times greatly excited her.

Another point to be borne in mind when studying blood pressure is that several readings, at an interval of perhaps a minute, should be taken before deciding what is normal pressure; as the excitement and pain due to the compression may produce a rather decided temporary rise.

Reducing by Increased Elimination.—Considering that hypertension in nephritis is due to retention of waste products, the most rational method of reducing it is to increase elimination through other channels, especially the bowels and skin. Free catharsis and gentle perspiration are safe measures, and may be employed even when the rise in pressure is only moderate. Even profuse sweating, if properly controlled, is a safe and efficient measure. Those foods the waste products of which are eliminated through the kidney should be restricted. This phase of the subject has already been considered under diet. In a series of cases the author has observed the effect of profuse sweats of twenty to thirty minutes on the blood pressure. The reduction varied from ten to twenty points. The subjective relief obtained by the sweats is apparently largely independent of the changes in pressure. The redistribution of blood is probably an important factor; the dilatation of the cutaneous vessels relieving the congestion of the splanchnics. This change is analogous to what takes place in the Nauheim baths, in which the calcium and sodium salts and the carbonic acid gas, by their irritating action, produce distinct hyperemia of the skin. After a sweat, a period of three or four hours may elapse before the pressure returns to the previous level. When daily sweats are given for a period of two weeks or more, a patient occasionally shows a permanent fall in pressure, it remaining below the original level even for a few weeks after the baths have been discontinued. One patient, where the pressure was taken at irregular intervals for three years, who had never been below 210, after five sweats remained for ten days below 185 mm. In this case, with the reduction in pressure, the patient suffered from repeated attacks of transitory hemiparesis with numbness. As he previously had been entirely free from attacks of this character, and they entirely disappeared soon after the sweats were discontinued, it is probable that they were due to the sweating. This can best be explained by assuming that the brain could only be sufficiently supplied with blood through the narrowed vessels when the pressure was very high. Lowering of the pressure led to cerebral

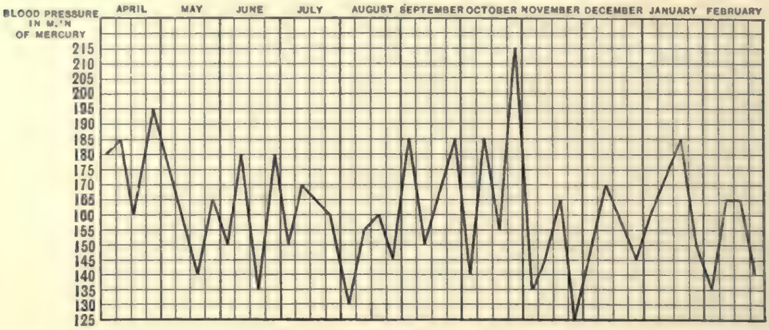

FIG. 1

ischemia with transitory hemiparesis. Disturbances of this character have also been observed after the use of vasodilators.

Venesection.—Butterman bled ten students, withdrawing, on an average, 300 c. c. (10 oz.). Nine had a fall in pressure varying from 5 to 30 mm. The author has noted the effect on blood pressure in five cases after withdrawing 500 c. c. of blood. No effect was observed in one case, the other four showing a reduction varying from 5 to 15 mm. The effect upon pressure is dependent largely upon the rapidity with which the blood is withdrawn, slow bleeding often having very little effect. The results of venesection are not sufficiently striking to warrant its use except in those patients who are full blooded. It would appear that much the same results, as far as reduction of pressure and removal of waste are concerned, can be accomplished by the sweating and without so severely taxing the patient.

Vasodilators.—Catharsis, sweating, bleeding, and rest may be considered rational measures for the reduction of pressure when due to retention of waste products. Reduction of pressure by the use of vasodilators is purely palliative, and, as has been mentioned, may do harm by lessening elimination through the kidney. Their use had best be restricted to those patients with threatened cardiac insufficiency, the result of hypertension, and to cases of extremely high blood pressure, where there is danger of hemorrhage from vascular injury. Sodium nitrite and nitroglycerin are the two vasodilators most frequently employed. Erythrol titranitrate has been less extensively used, and vasotonin has recently been introduced by Müller.

Nitroglycerin is best administered in the form of a 1 per cent. alcoholic solution. This preparation is much more reliable than the tablet form. There is considerable difference of opinion in regard to the dosage. Enormous doses, 0.3 gm. (5 grains), Stewart, von Noorden; 0.5 gm. (7½ grains), Hochbauss, have been given without ill effect. On the other hand, severe collapse has occurred after comparatively small doses, 0.5 mg. (1-120 grain), Loeb. There is evidently variation in the tolerance of different individuals. For the initial dose, 0.5 mg. (1-100 grain) is sufficient, if there is no effect on blood pressure within ten minutes, a second larger dose may be given. It can be frequently demonstrated that those patients who fail to react to moderate doses, 1 mg. (1-60 grain), rarely show a fall in pressure after larger amounts. When given on the tongue, the fall in pressure begins within two minutes, reaches its maximum in five to ten minutes, then the pressure gradually rises, until within thirty-five to forty minutes it has almost reached the original level. Nitroglycerin, when given to patients with extreme hypertension, does not always lower blood pressure. In a series of ten cases only seven reacted by a fall in pressure. In those susceptible to it the amount of fall in pressure varies greatly—20 to 50 mm. is about the average.

Sodium nitrite, on account of its prolonged action and absence of headache, is the most satisfactory vasodilator. The usual dose is 0.12 gm. (2 grains). When given in solution the effect appears within five minutes, reaches its maximum in from eight to fifteen minutes. The return to normal is more gradual than with nitroglycerin. Like nitroglycerin, not every patient with hypertension is affected by it. The average fall in pressure is about the same as with nitroglycerin.

Erythrol tetranitrate in 60-mg. (1-grain) doses produces a more marked reduction of the blood pressure than either nitroglycerin or sodium nitrate. After 1 grain, a patient of the author's reacted by a fall in pressure of 110 mm. The first effect appears within four minutes, reaches its maximum in about ten minutes, and this maximum effect is more prolonged than either of the foregoing. As with other vasodilators, about 30 per cent. of patients with hypertension fail to react. The chief objections to its use are its cost and the frequency with which it excites intense headaches.

The following chart shows the comparative effect of the various dilators on a series of ten patients:

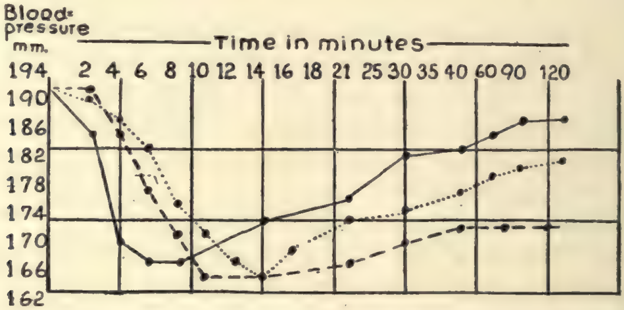

Fig. 2.—Comparative Effect on Blood-Pressure of Nitroglycerin (Solid Line), Erythrol Tetranitrates (Line Composed of Dashes) and Sodium Nitrate (Dotted Line).

Under the name of vasotonin, Mueller has prepared a combination of yohimbin—nitrate and urethan. This depresses blood pressure by dilating the peripheral vessels. Fellner reports that, when given subcutaneously, in man it reduces blood pressure without affecting the heart. The dose—10 mg. (1-6 grain)—is given every second day. The preparation is expensive, and more extensive clinical reports are necessary before definite statements can be made in regard to its value.

In the treatment of the headaches associated with high blood pressure the vasodilators are of little value. The various coal-tar products often give considerable relief, and in moderate amounts may be resorted to in case the pain is severe.

High frequency electricity has been recommended for the reduction of blood pressure. Arnold reports his results in 270 cases where the average fall in pressure was 12 mm. With the wide normal variations in pressure the question arises whether this change was really due to the treatment. More investigation of this method of treatment is advisable.

Summary.—Hypertension in nephritis is probably a compensatory process and should be treated by attempting to remove the cause. With our present knowledge, it appears that imperfect elimination through the kidney is an important element in its development. If this is true, the rational treatment is to restrict those foods the end products of which are eliminated through the kidney, viz., proteids; to increase elimination through the bowel and skin by free catharsis and sweats. Vasodilators relieve the condition temporarily without removing the cause and are chiefly indicated, and then in conjunction with the above-named measures, when the pressure becomes exceedingly high or on the appearance of evidence of cardiac incompensation. In many cases of extreme hypertension the ordinary vasodilator drugs fail to reduce pressure. In all cases the reduction in pressure is transitory in character. Sodium nitrate, on account of its more gradual effect, prolonged action, and absence of untoward symptoms, especially headaches, is the most satisfactory drug of this group.

Use of Diuretics.—Diuretics may be used in all forms of nephritis, provided we avoid those preparations which act as intense kidney irritants. Turpentine, cantharidin, juniper berries, and gin should be avoided not only in the acute, but also in the chronic, forms of nephritis. The rather common practice of the lay people to administer gin in cases of anuria is distinctly dangerous.

The various alkaline diuretics, as the citrate, acetate, and tartrates of sodium, potassium, and ammonia may be given. The nitrates may also be used, but are somewhat more irritating to the kidney. In nephritis with edema, however, alkaline diuretics usually fail to produce any diuretic action. If after a fair trial in these cases diuresis does not result, the diuretic should be discontinued. The acetate of ammonia, sodium, or potassium is probably most frequently prescribed. They may be given in gram (15-grain) doses in solution. Liquor ammonii acetatis, 10-25 c. c. (2-6 fl. drs.), or liquor ferri et ammonii acetatis, 15-30 c. c. (3-7 fl. drs.) are favorite preparations, the latter especially, as its iron content assists in combating the anemia. It is true that the ammonia is oxidized in the tissues and excreted as urea, thus increasing the work of the kidney. However, the amount is so small that it can be overlooked.

Caffein or theobromin-sodiosalicylate (diuretin), and theocin, an artificial theophyllin, are more powerful diuretics than the alkaline group. In addition to their effect on the kidney, they stimulate the heart, and thus assist in causing diuresis. Caffein was formerly thought to excite diuresis by direct stimulation of the renal epithelia. It has been demonstrated by Loewi and others that the diuresis is due to dilatation of the renal vessels and interference with reabsorption in the renal tubule. The ordinary dose of caffein is 0.15 gm. (2 grains). Better results may often be obtained by intramuscular injection of some soluble salt of caffein, as the salicylate or sodium benzoate. In place of the caffein, diuretin or theocin, the former in doses of 0.5-1 gm. (7-15 grains), the theocin in doses of 0.2-0.3 gm. (3-5 grains). The diuretin is soluble and is best given in solution. The theocin is quite insoluble, and may be given in powder or capsules. A very serious objection to all caffein preparations is their irritating action on the stomach and their tendency to produce insomnia. The gastric disturbance may be avoided by giving the caffein hypodermatically or in the form of rectal suppositories. As many of these patients already have difficulty in obtaining sufficient sleep on account of dyspnea or other causes, the additional insomnia caused by the caffein is a serious objection. Moreover, in severe forms of nephritis, with scant urine, caffein, like all other diuretics, usually fails. This restricts greatly its field of usefulness, and it is only rarely, in cases where anuria is threatening, that caffein can be relied upon.

Digitalis is another very valuable diuretic, and this action may be independent of its effect on the heart, as it has been shown by Jonescu and Loewi that the cause of the diuresis is a dilatation of the renal vessels due to a peripheral action and not to its cardiac effect. One objection urged against digitalis in nephritis is that it raises blood pressure. Cushney, in a recent Harvey lecture, calls attention to this fallacy, as in man it does not cause a constriction of the peripheral vessels or a rise in blood pressure. Indeed, in some cases it is distinctly lowered. This is due, according to Cushney, to the more perfect vasoconstrictor mechanism in man. With the increased systolic output following the use of digitalis, the vasomotor center receiving more blood, the tendency to increased blood pressure is overcome by a compensatory dilatation of the splanchnic vessels. Even after intravenous use of strophanthus the rise in blood pressure is negligible unless the individual blood pressure before the administration is normal (Vagt). Eychmuller has shown that this is also true after the intravenous administration of digalen. There is no reason why a patient with evidence of cardiac incompensation due to high blood pressure should not be given digitalis, and clinical experience has shown that the results are often very satisfactory. When given for its diuretic effect, it is a matter of indifference in what form the digitalis is given. The superior claims made for the infusion is chiefly due to the relatively larger dosage of this preparation. Recently preparations made from the posterior lobe of the hypophysis have been recommended as diuretics. Schäffer has shown that the pressor substance obtained from this portion of the hypophysis, when administered intravenously or subcutaneously to animals, excites marked diuresis. When given by mouth it is inactive. At the present time there are two preparations of hypophysis extract in the market—Pituitrin and an extract prepared by Burroughs and Welcome. The former can be given in the dose of 1 c. c. (15 min.), the latter is put on the market in ampules, each containing a single dose. These preparations cause a marked rise in blood pressure, and should not be used in patients with hypertension.

There is no evidence that the diuretics mentioned are harmful. The great difficulty is that in cases of renal edema they often fail to excite diuresis. When a diuretic fails to act after the lapse of a few days it is advisable to discontinue its use.

In the study of experimental nephritis some very interesting information has been acquired in regard to the effect of diuretics in acute nephritis. By employing different agents it is possible to produce in animals changes which are confined, early at least, chiefly either to the epithelium of the tubules, or the vascular apparatus, especially the glomerulus. Animals with the tubular type of nephritis respond to the action of diuretics. Later only, as the process extends and involves the vascular apparatus, do diuretics fail to act. Hedinger has shown that, in the early stages of this type of nephritis, caffein and digitalis give rise to unusually profuse diuresis. With the vascular type of experimental nephritis, from the beginning, diuretics are without effect, and no dilatation of the renal vessels can be demonstrated after their administration. This failure of response to diuretics is coincident with the appearance of the edema. In other words, where the kidney in nephritis reaches the point where it is unable to eliminate water, it also fails to respond to the action of diuretics. This is in direct accord with clinical experience, that diuretics often fail to relieve renal edema. More important in the treatment of edema are the restriction of sodium chlorid and fluids and increased elimination through the bowel and skin.

Summary.—The various alkaline diuretics, caffein, and digitalis may be safely administered in all forms of nephritis. Those diuretics directly irritating to the kidney, as turpentine, cantharidin, and gin, should be avoided. Animal experimentation and clinical experience have shown that when the kidney has lost its ability to eliminate water it has also lost its power to respond to diuretics.

Treatment of Uremia.—The rational treatment of uremia would be to increase the functioning power of the kidney. As it is only possible to accomplish this in a limited degree by the use of diuretics, our efforts should be directed toward restricting to a minimum the amount of waste to be eliminated through the kidney. This is accomplished by placing the patient upon a low proteid diet, and in case there is evidence of poor elimination of water to restrict fluids. The second important measure is to increase elimination through other channels, viz., bowel and skin.

In acute uremia the restriction of proteids may be carried to an extreme degree. With chronic uremia such restriction is useless. A certain amount of nitrogen—6 to 8 gms., equal to 35-50 gms. proteid—will be present in the urine, even in starvation. In a proteid-free diet this nitrogen arises from the destruction of body albumin. Lusk calls attention to the sparing influence of carbohydrates on protein metabolism. On an abundant carbohydrate diet, containing only 1 gram of nitrogen daily, Landgren was able to obtain a urinary nitrogen of only 4 grams, while on a starvation diet more than double this amount of nitrogen appeared in the urine. Fats protect metabolism to a less degree, although on a diet containing half its calories in carbohydrates and half in fats the protecting power to the protein is the same as on a pure carbohydrate diet. (Landgren.) This shows the importance of giving a patient on a low proteid diet abundance of carbohydrates and fats, and especially the former. Fifty grams of proteid daily is probably the minimum amount upon which a patient can be kept for a long period. Restriction beyond this point, except for short intervals, is not advisable. In the acute uremias, with gastric disturbance, the proteids should be given in an easily digested form, as milk. Provided we keep within the prescribed amount, there is no objection to the use of eggs. However, when we give proteid in such a concentrated form as meat or eggs, we are very apt to exceed the limit. For this reason, in acute uremias, our chief source of proteid supply should be milk, or, what is better, on account of its higher food value, cream. The patient with acute uremia may be safely kept for a week on a pint of cream daily. This can be given by diluting it with milk, seltzer, or vichy, so that the patient takes in this way about one quart of fluids daily. If the patient has marked edema, it is advisable to limit the daily fluid intake to this amount. Where there is anuria with very little edema an effort may be made to excite diuresis by free water drinking, subcutaneous transfusion with normal salt, or with normal salt solution by rectum. If the kidney reacts to the increased fluid intake, it should be continued. Where diuresis does not follow such measures, and this is the rule, they should not be continued, as they will only increase the edema. In addition to the cream, there can be no objection to the use of carbohydrates. Sugar may be given freely, provided the patient will take it, in the form of sweet lemonade, jellies, honey, etc. Cereals and bread are harmless, and these may be used as a vehicle for the cream. In fact, any of the carbohydrates are allowable. As a rule, however, in acute uremia the patient's appetite is much impaired, or he is comatose, so that it becomes very difficult to give food. For this reason it is usually necessary to restrict his diet to liquids, and for this purpose the cream is the most suitable. If improvement occurs, carbohydrates, as cereals, bread, sugar, jellies, etc., may be given in order to increase the caloric value of the diet.

Patients with threatened uremia should be placed upon a low proteid diet. One pint of 20 per cent. cream (equal to 1,000 calories) contains about 18 grams of proteid. One quart of milk (equal to 650 calories) contains about 30 grams of proteid. This shows the advantage of the cream over the milk. In addition, the patient may receive various carbohydrates, bread, cereals, cooked fruits, jellies, etc. When necessary, these foods may be used as a vehicle for the cream. The quantity of cream may be increased if necessary to 1½, or even 2, pints (1,000 c. c.) without increasing the amount of proteid beyond the permissible quantity. The fluid intake in threatened uremia depends upon the permeability of the kidney to water. If this is good, abundant water may be advantageously given. If edema is present, indicating inability of the kidney to secrete water, then the amount of fluid should be restricted. When fluids are indicated, there is nothing better than good plain water.

Free elimination through the bowels is desirable. For this purpose the saline cathartics are most commonly employed. Where the patient objects to these, compound jalap powder, elaterin, phenolphthalein, or any other laxative that will produce a watery movement.

Elimination through the skin by means of vigorous sweats once or even twice a day is of great value and should be attempted in all cases of acute uremia. Rarely is a patient's heart so weak that he cannot safely take a good sweat, if properly administered and supervised. The method of giving the sweats has already been considered. Pilocarpin may be used to produce diaphoresis, but is not as desirable as heat. Venesection may be of decided value, especially in well-nourished, full-blooded persons. It is questionable whether it has many advantages over sweating, still in urgent cases 500 to 750 c. c. (17-25 oz.) of blood may be withdrawn from one of the superficial veins by cutting down on the vein, or by introducing into it an aspirating needle, and allowing the blood to escape. If this last method is employed and the needle tends to become obstructed, the clot may be removed by using a little suction. If the patient is not edematous, following the venesection, he may be transfused with ½ liter of normal salt solution. Especially in comatose patients, venesection and transfusion may give decided relief.

The various diuretics, especially the caffein preparations and digitalis, may be tried. On account of the nausea and the tendency of caffein to irritate the stomach, it is best given hypodermatically, using some soluble salts like caffein sodium, salicylate, or benzoate, 0.15-0.2 gm. (2-3 grains) every four hours. Any reliable preparation of digitalis may be used. It is especially desirable where the uremia is associated with weak heart action, but even in cases with very high blood pressure it may be safely tried. The very high blood pressure may be somewhat lowered by the various vasodilators, of which sodium nitrate is the most satisfactory.

When the patient is edematous, drainage by incisions or puncture of the legs is indicated. The draining of such accumulations is always advisable, as it avoids the necessity of reabsorption into the blood of material which is supposedly toxic. The relief obtained by draining from the extremities often enables the patient to rest more comfortably and perhaps obtain some needed sleep. If the body cavities contain any considerable amount of fluid, it should be aspirated.

It is exceedingly difficult to relieve the headache of which many uremics complain. Increased elimination is most essential. Nitroglycerin or sodium nitrate rarely give relief, and not infrequently increase the pain. Many of these patients with severe headaches find of their own accord that certain headache powders give them considerable relief. Where the pain is intense the coal-tar products may be safely administered. They tend to lower blood pressure, although it can be demonstrated that patients with hypertension who obtain relief from their headaches by taking coal-tar products frequently do not have a reduction in pressure when the pain disappears.

The uremic vomiting is rarely benefited by the use of bismuth or other gastric sedatives. If the patient's condition warrants it, lavage of the stomach frequently gives temporary relief. It is dangerous, however, to attempt to pass the stomach tube if the blood pressure is very high or the patient's heart action poor. Drinking large amounts of water may wash out the stomach and give temporary relief. In many of these cases the vomiting is central in origin, and any local measures are of little value. Where the vomiting is intense and persistent the question may arise as to the advisability of using morphin. The chief objection to its use is that it constipates and lessens the secretions from the skin, and also probably the secretion of urine. In spite of these objections there are occasions when relief from the vomiting becomes imperative on account of exhaustion and a hypodermic of morphin, 8-16 mg. ($\frac{1}{8}$-$\frac{1}{4}$ grain), may be given.

To control the extreme restlessness, 0.7 gm. (10 grains) of chloral may be given by mouth, or in case the patient vomits 2-3 grams (30 to 45 grains) per rectum. Morphin is not desirable, but may be given if other measures fail to control the restlessness or vomiting. As the vomiting is probably usually of cerebral origin, local measures to the stomach rarely are of much value.

Uremic Dyspnea and Pulmonary Dyspnea.—The dyspnea associated with renal diseases is usually of cardiac origin. Rarely is there a real bronchial asthma. So-called uremic dyspnea is almost invariably associated with hypertension and not infrequently passes into pulmonary edema, as both are due to the same causative factors. As a result of the high blood pressure, the left ventricle is unable to completely empty during systole. When this residual blood has accumulated until there is insufficient room in the left ventricle for the volume received with each systole from the right heart, there follows hyperemia of the pulmonary vein and artery, the lung becomes more voluminous, and its elasticity is impaired. Proper aeration of the blood is interfered with, partly from lessened flow of blood through the lung, and partly from the hyperemia with lessening of the air space. If this pulmonary stasis is not relieved, the pressure in the pulmonary artery increases and, with this, pulmonary edema develops. Adrenalin, when injected intravenously into a rabbit, not infrequently gives rise to acute pulmonary edema. If a cannula connected with a mercury manometer is placed in both carotid and pulmonary artery and the heart exposed, it will be noted, after the adrenalin is given, that there is a sudden marked rise in blood pressure. Now, if the left ventricle is unable to empty completely against this increased resistance, it dilates, and at this time the pulmonary pressure begins to rise; the right ventricle dilates as evidence of increased work on the right heart due to backing up of the blood from the overfilled left ventricle, and at this point pulmonary edema develops. High blood pressure, however, is not the only cause of acute pulmonary edema. This same disproportion between the working power of the two sides of the heart may arise where there is a weak left ventricle, without high blood pressure; dilatation and stasis then occur. It has been the author's experience, however, that acute pulmonary edema in nephritis is confined to those patients with hypertension. The origin of uremic dyspnea and pulmonary edema has been given in some detail, as it gives us a clue to the method of treatment.

When associated with high blood pressure an effort should be made to relieve the left heart. This may be accomplished by a vasodilator or bleeding. Second, to stimulate the heart, and this can best be done by intravenous injections of digalen or strophanthin. When experimental acute pulmonary edema has been produced in an animal by using adrenalin, the best results are obtained by bleeding from an artery, and at the same time injecting digitalis intravenously. By this combination the balance of the circulation is restored. When the pulmonary edema is due primarily to weakness of the left ventricle, without high pressure, venesection, with cardiac stimulation, fulfils the indication. Where the edema is associated with high blood pressure bleeding gives better results than the vasodilators. In all cases of acute pulmonary edema in man morphin is indicated. Relief is obtained by its use more frequently than by other means. Its method of action is not clear. Perhaps part of the benefit is due to allaying the patient's fear. Atropin has been recommended and, apparently in some cases, gives relief. Lead acetate, at one time highly recommended, is now rarely used. Adrenalin has been recommended in the treatment of acute pulmonary edema. In all cases, if given in a manner that is followed by its physiological effect, viz., intravenously or subcutaneously, adrenalin is contraindicated. Where the pulmonary edema is due to hypertension reduction of pressure is indicated. When due to primary weak heart cardiac stimulation fulfils the indication. Plenty of fresh air apparently is as beneficial as oxygen inhalation. To prevent attacks of acute pulmonary edema, the patient should avoid overexertion. Not infrequently such attacks are due to overtaxation, either physical or mental. Sweats and good elimination through the bowel are also of value in warding off attacks.

SUMMARY.—So-called uremic dyspnea and acute pulmonary edema are different stages of the same process. To avoid overtaxation, physical and mental, and to maintain good elimination are the best prophylactic measures. During an attack of acute pulmonary edema with high arterial tension the indications are met by bleeding and at the same time injecting slowly intravenously some digitalis preparation, as digipuratum. If it is thought inadvisable to bleed, use vasodilators, as nitroglycerin or sodium nitrate, and intravenous injections of digitalis. When the arterial tension is low, and there is evidence of venous stasis, perform venesection and intravenous injection of a suitable digitalis preparation. Morphin in moderate doses, 10 to 15 mg. (1-6 to ¼ grain) hypodermatically is safe, and often gives decided relief. Atropin and lead acetate are rarely beneficial. It is well to remember that attacks of acute pulmonary edema disappear, as a rule, within one or two hours even when untreated.

Attacks of acute pulmonary edema very frequently develop just after retiring. To ward off these attacks the patient should be advised to undress slowly, to have the room and bed comfortably warm, and to take, before undressing, 2 grains of sodium nitrate. If, in spite of these measures, the attacks persist, the patient should remain in bed for a week or more in order to rest the heart. To avoid attacks of acute pulmonary edema rest is most essential.

SPECIAL TREATMENT

ACUTE NEPHRITIS

In acute nephritis there are active inflammatory or degenerative changes in the vascular, interstitial, or epithelial structures in the kidney. While the most active change may be confined to one of these structures, all are usually more or less extensively involved, especially after the process has continued for some time. The treatment consists in prophylactic measures to prevent or modify the intensity of the inflammatory or degenerative changes, and the active treatment of the fully developed nephritis.

Prophylaxis.—The chief etiological factors in acute nephritis have already been mentioned and briefly are acute or chronic infections, chemical agents, cold, and products of disturbed metabolism. Scarlet fever and diphtheria are the two acute infections most frequently complicated by nephritis. Chronic infections much less frequently cause trouble, although tuberculosis or syphilis is frequently responsible for a mild grade of acute nephritis. Acute nephritis not infrequently follows very mild acute infections, especially tonsillitis. Complete removal of the tonsils is advisable in children subject to repeated tonsillar infection, not merely to avoid nephritis, but to ward off attacks of acute articular rheumatism. Gargles and sprays are probably of little value as preventive measures, as usually the infection begins deep down in the crypts, far beyond the reach of local agents. Any tonsil, when associated with cervical glandular enlargement, should be removed.

It is questionable how much may be done in scarlet fever to prevent nephritis. The determining factors are probably the intensity of the infection and the individual susceptibility. At the present we do not possess any efficient measures for combating either the intensity or duration of the toxemia. Good elimination through the bowel, kidneys, and skin may assist in lessening the toxemia. This may be accomplished by cathartics, free fluid intake, and warmth. On account of the importance of cold in nephritis, the question arises as to the advisability of cold baths in the acute fevers, especially scarlatina. There is no positive evidence that patients so treated are more subject to nephritis. It is advisable, however, to avoid the very cold bath. Rest in bed during the acute infection, by maintaining uniform temperature of the skin and reducing body waste, lessens the amount of kidney work. The early recognition of diphtheria and prompt administration of antitoxin lessen the probability of nephritis.

In diseases that predispose to nephritis kidney irritants should be avoided. This includes salol, salicylates, potassium chlorate, mercury, especially bichlorid, turpentine, copaiba, cubebs, and cantharides. The condiments and sharp vegetables, like onions and radishes, have the reputation of being kidney irritants and, until their action has been definitely determined, should be avoided. Strong alcoholic drinks should not be used, as they undoubtedly irritate the kidney. Moderate amounts of alcohol probably do not exert any injurious effects, but in those acute infections, which are prone to excite acute nephritis, it is safer to avoid alcohol entirely.

Active Treatment.—There is no specific therapy in nephritis. One's efforts should be directed, first, to limit extension of the process. Second, to put the minimum amount of work on the kidney by restriction of diet, and increased functioning of the other organs capable of assuming, in part, the work of the kidneys; third, the treatment of symptoms that arise, as edema, uremia, etc. Known kidney irritants in the form of foods should be avoided. Rest in bed is imperative, as it reduces to a minimum the body waste and offers the best protection against chilling of the skin. The body should be kept as warm as is consistent with the patient's comfort. Placed between warm woolen blankets, the patient should be allowed an abundance of fresh air. The mistake is frequently made of inducing heat by raising the room temperature to an uncomfortable degree. This is not only unnecessary but harmful. The skin should be thoroughly cleansed daily with soap and tepid water. Cold baths, except when necessary to reduce temperature, should be avoided. Extreme cold should be avoided, as a tepid bath with good friction will usually reduce the temperature. Cocoa butter and other oily preparations should not be applied to the skin, as they interfere with perspiration.

Catharsis.—The bowels should be kept free, the degree of catharsis depending upon the intensity of the intoxication and amount of edema. It is not advisable to resort to prolonged, extreme catharsis on account of the resulting emaciation. Laxatives producing a watery movement are most desirable. Magnesium or sodium sulphate in one-half-ounce (15-gm.) doses, in half a glass of hot water before breakfast, is most frequently employed and, when well borne by the patient, is entirely satisfactory. Sodium phosphate in half-ounce (15-gm.) doses in hot water is somewhat more palatable. Seidlitz powder may be preferred by some. The natural or prepared saline waters, provided they do not contain large amounts of sodium chlorid, make a rather agreeable form for administering the saline. Effervescing preparations are often more palatable—liquor magnesii citratis, 12 fl. oz. (360 c. c.), lithii citras effervescens, 1-2 oz. (4-8 gms.), magnesii sulphas effervescens, 1-4 oz. (4-32 gms.). When the stomach is disturbed calcined magnesia (magnesia oxidum), 5-60 grains (0.3-4 gms.), is often well borne. Calomel is commonly used, but except for its slight diuretic action it does not possess any special advantages. Senna, cascara, rhubarb, aloin, elaterin, or any of the other vegetable cathartics may be tried. Elaterin, gr. 1-16 to 1-10 (4 to 6 mg.), gives a very watery movement, and is especially valuable in extensive edema, or threatened uremia. Another favorite laxative, although disagreeable to take, is compound jalap powder in dose of 15-60 grains (1-4 gms.). When the patient is delirious or comatose, one or two drops of croton oil on sugar may be given. Enemas rarely give a sufficient movement, but may be advantageous when combined with laxatives.

Diuretics.—Diuretics may be tried to increase the flow of urine, but are rarely of value in acute nephritis. It has been shown experimentally that when a kidney fails to respond to simple diuretics like water it rarely responds to the more powerful ones. The acetates, citrates, or nitrates may be tried in doses of 15 grains (1 gm.) dissolved in water. There is no objection to trying caffein, 2 grains (0.12 gm.), theobromin sodium salicylate (diuretin) 10-15 grains (0.7-1 gm.), theophyllin

3-5 grains (0.2-0.35 gm.), dimethyl xanthin (theocin) 3-5 grains (0.2-0.35 gm.).

It was formerly urged that caffein preparations irritated the kidney and were contraindicated in acute nephritis. This has recently been disproved. All caffein preparations are gastric irritants and tend to produce insomnia. For this reason their continued use is not advisable. A safe rule to follow in acute nephritis where diuretics are indicated is to determine the daily urinary output first, then try the various diuretics and determine if the output of water is increased. By following this method it can be decided whether in the individual case a diuretic effect is obtained, and thus avoid the useless administration of drugs. When the heart action is impaired digitalis often acts better than any other diuretic. Under its use the urinary output is increased and the edema subsides. It is largely a matter of indifference what form of digitalis is used, as the results are dependent upon the activity of the preparation. When we are sure of a fresh leaf the infusion is a very satisfactory preparation. In this country, however, where the average pharmacist pays little or no attention to the freshness of the leaves, it is better to use a tincture prepared by some reliable manufacturer. It is now possible to purchase abroad a carefully prepared titrated tincture. Digipuratum is another reliable preparation. It is put on the market in tablet form, each tablet equivalent to 1½ grains of the dried leaf. High blood pressure, provided there is no evidence of cardiac incompetency, is not a contraindication to the use of digitalis.

Diet.—The chief indication in acute nephritis is to avoid foods that irritate the kidney, that throw increased work upon the kidney, or are poorly eliminated through the kidney. When dieting is continued for more than a few days it is necessary to consider the caloric value of the food. Substances that irritate the kidney have already been considered. They include condiments and certain vegetables, as onions, radishes, celery, garlic. According to von Noorden, asparagus is not included in this list. Orange or lemon juice is harmless. There is no objection to moderate amounts of tea, coffee, or chocolate. According to von Noorden, the following substances are excreted with difficulty through the kidney: urea, creatinin, pigments, hippuric acid, phosphates, chlorids, and usually water. On account of the poor elimination of urea the proteid in the food should be reduced to the minimum consistent with protecting the body albumin. On a proteid-free diet the body muscle is used, and a certain amount of nitrogen continues to be eliminated through the kidney. It is of advantage, therefore, to give sufficient proteid to prevent muscle destruction. This amount may roughly be considered as 50 gms. or about the amount contained in a quart and one-third of milk. Such low proteid consumption is only permissible for short periods of time, one or two weeks; for prolonged periods 60 to 80 gms. daily is needed. It can be readily seen that it is possible to administer excessive proteid on a pure milk diet as the patient receiving three quarts daily would be taking about 120 gms. of proteid daily, and to get rid of the consequent nitrogen waste must eliminate 30 to 35 gms. of urea daily. This far exceeds the ability of the acutely diseased kidney. It is not advisable to give the patient more than one quart of milk daily. To increase its food value one half a pint of cream may be added. Where the edema is very marked it may be advisable to reduce the fluids below this point and the patient may be given a total of one quart composed of equal parts of milk and cream, or even one pint of cream daily. While milk is a very desirable food it is not necessary to confine the patient with acute nephritis to this alone. Easily digested carbohydrates as cereals, toast, light desserts, cooked fruits, fruit jellies; and some of the green vegetables, as string beans, asparagus, and spinach, make a pleasant variety and add considerably to the food value of the diet. The addition of carbohydrates to the dietary becomes necessary when we wish to keep a patient for a prolonged period of time on a low proteid diet. Otherwise, emaciation results. One and one half quarts of milk contain 60 gms. of proteid, an excessive quantity for an acute nephritic; but the patient receives only a little over 1,000 calories daily or less than half the requisite amount. The additional 1,000 calories must be made up with foods rich in carbohydrates and fats. Abundance of sugar may be added to all the foods; in addition sweet lemonade, fruit jellies, honey, etc. Sugar has a high caloric value and is free from waste eliminated through the kidney. The addition of cream to the cereals and cooked fruits still further increases their food value. Milk and cream may be given in the form of cream soups, ice cream, milk chocolate, and in a variety of other forms to suit the patient's taste.

Creatinin, like urea, is not easily eliminated through the kidney. A considerable portion of the creatinin present in the urine arises from the destruction of body muscle. The chief source of ingested creatinin is meat extracts and meat broths. These should both be avoided in acute nephritis as, in addition to the creatinin, they contain rather large amounts of sodium chlorid and have a very low nutritional value.

Urinary pigment, phosphates, and hippuric acid are also eliminated with difficulty. The urinary pigments are derived almost entirely from the blood and, by omitting meat, we remove the main source of external supply. There is no means of reducing the amount of pigment derived from the normal destruction of red blood corpuscles. The chief course of phosphoric acid is meat, milk, yolk of eggs, and certain vegetables. In the ordinary diet of acute nephritis the chief supply comes from the milk. If we consider the amount of phosphates ingested in this way as harmful calcium carbonate may be given, as recommended by von Noorden. The calcium combines in the intestine with the phosphoric acid and renders it less readily soluble. Hippuric acid is formed in the kidney from benzoic acid. The amount of benzoic acid in the foods is so small that it is practically negligible.

Sodium Chlorid.—Attention has already been called to the importance of sodium chlorid restriction in nephritis. It is especially in the acute forms with edema that the beneficial results of its restriction are most marked. There is a sufficient amount of sodium chlorid in a quart of milk to supply the body requirement; another argument against the use of large quantities of milk in acute nephritis. No salt should be added to any of the food in acute nephritis with edema. Some advise the use of unsalted bread and butter. This may be advisable in extreme cases. Salted butter contains on the average 1 per cent. of salt, and bread 0.5 to 0.7 per cent. As all the fruits and carbohydrates contain very small amounts of salt they may be used freely.

Fluid intake in acute nephritis has been a subject of considerable discussion. Not every case of acute nephritis shows an inability to eliminate water. The presence of dropsy, however, is evidence of kidney impermeability to fluids, and taking large amounts of liquids only increases the edema. Where the kidney is permeable to water, as shown by the absence of edema, free drinking of liquids (and there is nothing better than plain water) increases the urinary output, not only of water, but also of solids; and flushes out the débris from the urinary tubules. It is scarcely necessary to state that the various waters recommended for the cure of nephritis do not possess any virtues over pure plain water; and some of them contain ingredients that are actually harmful to the kidney. Where the edema is marked the fluid intake should be restricted to the minimum; the patient's comfort being the only limitation. As regards fluids, each patient must be treated individually, being guided by the permeability of the kidney.

SWEATING.—To increase elimination of solids and water sweating is often desirable. The most marked benefit is noted in those patients with marked edema. It is possible with a good sweat to remove a quart of water and 15 grains or more each of urea and sodium chlorid. In addition to the eliminated matter some benefit may be derived from the effect on the circulation, the peripheral vessels being dilated. Whether sweating ever increases the degree of intoxication, also the manner of giving the sweat, have already been considered in detail under the general principles of treatment. Heat is the safest and most satisfactory diaphoretic.

DECAPSULATION, as introduced by Edebohls in 1901, has been largely abandoned. More careful clinical observations following the operation has failed to show that marked improvement resulted. Experimentally it has also been demonstrated that increased hyperemia of the kidney, as claimed by Edebohls, does not develop as a result of the operation. Although the operation is little resorted to at present occasional reports show that in some severe cases improvement apparently follows, and it may be in a few instances the operation is beneficial. It is not, however, free from danger, and it is questionable whether it should ever be advised.

Edema.—The edema can best be controlled by restriction of fluids and salts, free catharsis, sweating, diuretics, and direct removal, by tapping or drainage. Where the edema is marked, the fluid may be safely restricted for a few days to 1½ pints, this consisting of equal parts of cream and milk. This also supplies the patient with a low sodium chlorid diet. The subject of catharsis and sweating has already been considered. The alkaline diuretics, caffein preparations, or digitalis may be tried, but should be discontinued in case they fail to increase the urinary output. When fluid accumulates in considerable quantities in the body cavities, it should be withdrawn. When other measures have failed, incisions in the limbs or Southey's tubes may give great relief.

Anuria.—Anuria is usually associated with edema. The kidney has temporarily become completely impermeable to water. Increasing the fluids rarely excites diuresis. Efforts must be made to increase elimination through other channels, until the kidney function is regained. Catharsis and sweating, combined with low proteid diet and reduction of fluids, offer the best hope of relief. To relieve the thirst, ice may be sucked or water given in tablespoonful doses. Severe anuria is of short duration, the patient either dying or the urinary output increasing within a few days. Cream, cereals, bread, cooked jellies should constitute the diet. On account of the short duration of the anuria, extreme restriction in diet may be resorted to.

Uremia.—The probability of uremia must be considered in every case of acute nephritis, and for this reason prophylactic measures should be instituted from the onset of the trouble. These measures have already been considered, viz., low proteid diet and good elimination through skin and bowel. When uremia has actually developed, these measures must be pushed to the limit. When the edema is marked, aspiration of the body cavities and drainage of the legs are indicated. By these measures considerable quantities of more or less toxic material are removed. Removal of fluid in this manner is to be preferred to bleeding, as such fluid contains practically the same amount of toxic material as the blood. Transfusion, with the objections to increased fluid intake and excessive sodium chlorid, is contraindicated in uremia with edema.

The kidney is impermeable to water and transfusion merely increases the dropsy. When uremia develops without edema transfusion may be of value. The normal salt may be introduced intravenously (1 quart) or subcutaneously in the same amount. If marked diuresis follows, the transfusion may be repeated every four hours until the intoxication is alleviated. When diuresis does not result, it is inadvisable to repeat the transfusion. The introduction of normal salt per rectum by the drop method may be resorted to, and eight or ten quarts introduced daily, only, however, in patients free from edema. As a rule, however, patients with uremia from acute nephritis have marked edema.

Dyspnea.—Dyspnea may be a very distressing symptom in acute nephritis. It is, however, much less frequent than in the terminal stages of the chronic interstitial type. It is most frequently of cardiac origin. Digitalis may be tried. Withdrawal of fluid from the body cavities is often beneficial. Not infrequently a good sweat in the evening may give sufficient relief to enable the patient to obtain a night's sleep. When the dyspnea interferes with sleep, no drug gives as much relief as some form of opium. Although it constipates and lessens the secretion from the skin, it is nevertheless the safest remedy we can employ to induce sleep. With moderate doses, the secretion from the kidney is not affected.

Itching.—For the itching of the skin, so common in uremia, alkaline baths often give relief. We must be careful to avoid local applications that might irritate the kidney.

Backache, which is quite common in acute nephritis, is often relieved by local heat—a hot-water bag, or, still better, an electric pad. Vigorous massage of the lumbar region may give considerable relief. Simple plasters at times are beneficial, probably largely due to retaining the heat.

Edema of the glottis may be treated by scarification of the edematous area and in severe cases tracheotomy may be necessary. Local cold to the neck may be safely tried. For the pulmonary edema morphin is the most satisfactory remedy. When the patient is not very anemic, and the blood pressure high, withdrawal of a pint of blood may be beneficial. When the blood pressure is low the continued use of digitalis often controls the attacks. The subcutaneous injection of 20 minims (1.3 c. c.) of camphorated oil, or the administration of aromatic spirits of ammonia ʒj (4 c. c.), during an attack may have sufficient effect upon the heart to correct the disproportion between the working power of the left and right ventricle, the usual cause of acute pulmonary edema. Oxygen inhalations are also beneficial. Lead acetate, as recommended by Traube, has never given any results in my hands. Atropin rarely is of any benefit, and when the pulmonary edema is associated with high blood pressure may do harm.

Headache.—Local heat or cold may be used for the relief of headache. In case the heart action is good, some of the coal-tar products may be used. Headaches are usually symptoms of intoxication, and increased elimination is indicated.

Anemia.—For the anemia some simple form of iron, as the Blaud's pill, is satisfactory.

Gastric Disturbances.—These are uremic in origin, and local measures rarely give relief, although persistent lavage often is temporarily beneficial. Gastric sedatives are of little value. When nausea appears all drugs which might act as gastric irritants should be discontinued.

HEMORRHAGIC NEPHRITIS does not require any special treatment. Ergot, suprarenal extracts, and the various styptics do no good, and may derange the gastrointestinal tract. Calcium lactate, grains 15 (1 gm.), three times daily, may be used if the blood coagulability is disturbed.

Summary.—In acute nephritis the patient should be placed upon a low proteid diet, not to exceed 50 grams daily. This amount is contained in 1½ quarts of milk. To increase the food value of the diet carbohydrates may be safely given. Cream is also valuable. When edema is present salt and fluids should be restricted. Good elimination through the skin and bowel is most important. Diuretics may be tried, but usually fail to act. Edema may be relieved by aspiration of the body cavities, or drainage from the legs by incision. When uremia is impending, elimination through skin and bowel should be augmented, and the proteid content of the diet further restricted, by placing the patient upon one pint of cream daily, combined with simple carbohydrates.

CHRONIC PARENCHYMATOUS NEPHRITIS

An acute nephritis may pass into a chronic form. More frequently, however, chronic parenchymatous nephritis begins as such. All of the etiological factors responsible for acute nephritis may also give rise to the chronic form. The more important of these are acute or chronic infections, lead, mercury, alcohol, autointoxication, the result of chronic constipation, certain metabolic diseases, as gout and diabetes mellitus. The disease is more prevalent in cold or rather changeable climates, and chilling, no doubt, plays a rôle. The treatment of complications, as edema and uremia, has already been considered in the treatment of acute nephritis. The principles of treatment throughout are the same as those employed in the acute forms. The main differences are that here we are dealing with a chronic disease and the patient's method of living, as regards exercise and diet, must be so arranged as to be consistent with years of life. It is a disease characterized by repeated acute exacerbations, each of which leaves the kidney more impaired. During these attacks the treatment is the same as outlined in acute nephritis. Excessive muscular exertion, acute infections, chilling, errors in diet and intestinal intoxication are most frequently responsible for the acute exacerbations. Drugs that might irritate the kidney must be avoided. The degree of rest desired will depend upon the intensity of the process. Patients with slight uremic symptoms or marked edema belong in bed, or at least at rest. Patients with no evidence of the trouble, except the urinary findings, should be advised to avoid excessive muscular exertion and exposure to wet cold. It is advisable in even the very mild types to select a climate where the winters are mild. California is a very good all-year climate.

Elimination.—Good elimination through skin and bowel is important. Even the mild cases should take a daily warm bath. The degree of catharsis required depends upon the intensity of the process.

Diuretics.—Diuretics usually have the desired physiological effect in chronic parenchymatous nephritis. When the edema is marked, caffein preparations or digitalis may be used. In the milder forms an alkaline diuretic, as liquor ferri et ammonii acetatis, ʒiv (16 c. c.), three times daily, possesses the additional advantage of supplying iron. As all the severe cases are decidedly anemic, iron in some form is needed.

Diet.—Diet is one of the essential parts of the treatment. The diet should be free of substances that might irritate the kidney, contain merely enough protein to supply the body needs, and possess the proper caloric value. The sharp vegetables, alcoholics, condiments, and meat soups containing considerable meat extractives, should be avoided. The amount of proteid ingested is dependent partly upon the severity of the attack. Although there is considerable difference of opinion as to the minimum amount of proteid consistent with good health, 80 gms. daily may be taken as a fair average. On an average about 20 gms. of proteid is taken daily in the form of vegetable albumin. One quart of milk contains 40 gms.; two eggs, 12 gms. This would leave only 8 gms. to be taken in the form of meat proteid, equivalent to 48 gms., or a little more than 1½ ounces of meat. It is a matter probably of little importance in what form the albumin is taken, whether meat, eggs, or milk. For that reason more meat may be given and less milk. It is more important that the total daily amount does not exceed the body requirement. It has been claimed that the extractives in meat render it less desirable than milk. There is no good evidence to support this, as in von Noorden's clinic patients with chronic nephritis did quite as well when given their albumin in the form of meat as when taken as milk. When meat is taken, it is a matter of indifference whether it is light or dark. Wild game is not desirable on account of the danger of decomposition having set in, as food of this sort does not receive the proper care after killing. Smoked meats are considered objectionable on account of possible irritation of the kidneys. Sausage, as it contains a great deal of pepper, is also objectionable. Eggs may be taken in any form desired. It is only necessary to restrict the patient in his proteid foods. The fats and carbohydrates may be taken freely, avoiding certain irritating forms already mentioned. Well-cooked green vegetables, root vegetables, cereals, fruits, and the various simple desserts may be safely given. Sugar may be given freely, as it has a high food value and is free from nitrogenous waste. The above mixed diet is not merely permissible, but it has distinct advantages over the pure milk diet. If milk alone is used, at least three quarts must be taken daily. This contains 120 gms. of albumin, far in excess of the needed amount, thus overtaxing the kidney. Furthermore, this gives an excessive amount of fluid.

Restriction of Fluids.—The restriction of fluids as recommended by von Noorden in certain cases of nephritis has been too generally applied. It is necessary to determine in every instance the permeability of the kidney to water. If this is good, then the patient should receive sufficient fluids to bring the output of the urine up to 1½ quarts in the twenty-four hours. In addition, once or twice a week, the patient may receive a flushing-out day when he receives fluids sufficient to yield two quarts or more of urine daily. This assures good elimination of nitrogenous waste. As patients with chronic parenchymatous nephritis rarely have high blood pressure, it is not necessary to restrict fluids in order to protect the heart. When the kidney is impermeable to water, as shown by the presence of marked edema, which increases with free drinking of fluid, excessive water drinking only does harm. In these cases the bowel and skin must temporarily act vicariously for the kidney, as we cannot hope to eliminate the requisite amount of urinary waste through the natural channel.

Sodium Chlorid.—Comparatively simple rules can be established in regard to the use of table salt. Every healthy individual consumes many times the amount of sodium chlorid demanded by the body. This excess is eliminated largely through the kidney. In many cases of chronic parenchymatous nephritis the kidney is impermeable to salt. When taken in excess of the daily requirement it remains in the body and retains with it sufficient water to maintain the proper molecular concentration. This is the basis of Widal's theory of edema. It is advisable in every case of nephritis with edema to reduce the sodium chlorid to about 2 gms. daily. This is about the amount contained in one quart of milk. No salt should be added to any of the foods, except the small amount used in bread. Clear soup, which contains much salt, should be avoided. With every precaution to restrict salt, there is no danger of giving the patient an insufficient amount. Some advise carrying the restriction to the point of using unsalted butter. As the use of excessive amounts of salt is a habit, the patient soon becomes accustomed to the lessened quantity.

Tea, Coffee, Tobacco.—There is no objection, as far as the kidney condition is concerned, for the patient to use moderate amounts of tea or coffee. When insomnia exists the use of coffee should be restricted. Taken to excess, coffee acts as a cardiac irritant. Moderate smoking probably does little, if any, harm, as, with tea and coffee, it is the excessive use that is injurious. To deprive the patient with chronic parenchymatous nephritis of a cigar after each meal is, with our present knowledge of the action of tobacco, unwarranted.

Where we have a mixed form of nephritis, or when a chronic parenchymatous nephritis passes into a secondarily contracted kidney, the blood pressure may be increased, and it becomes necessary to consider the proper treatment of the symptoms. This will be considered under the treatment of chronic interstitial nephritis.

Summary.—The treatment of chronic parenchymatous nephritis is in general the same as the treatment of acute nephritis. The chief point of difference is in the diet, as we are dealing with a chronic process and extreme restriction of proteid is not feasible. Foods or drinks known to be renal irritants should be avoided. Carbohydrates and fats may be taken freely; the proteids should be restricted to 80 or at most 100 gms. daily. It is probably a matter of little importance in what form the proteid is taken, whether in milk, meat, or eggs. In the presence of edema water drinking should be restricted. The salt should be restricted to that naturally present in the food. Edema is to be treated by restriction of fluids and salt, catharsis, sweats, diuretics, withdrawal of fluid from the body cavities, and, if necessary, incisions in the legs. Iron preparations and special foods for the anemia. Uremia is to be treated by good elimination and restriction of proteid.

Chronic Interstitial Nephritis

The treatment of chronic interstitial nephritis is the same whether it be of the primary or secondary type. The only essential difference in the treatment and that of chronic parenchymatous nephritis is in the management of the high blood pressure. Edema, when of renal origin, and uremic symptoms are treated in the same manner in all forms of nephritis. Probably every patient with chronic interstitial nephritis and compensated heart has an increased blood pressure. As a rule, it is far above normal, not infrequently reaching 220 mm. of mercury. This throws an enormously increased amount of work upon the heart, which, sooner or later, succumbs to the strain. The main object of the treatment is, therefore, to lower blood pressure and protect the heart. This increased blood pressure frequently results in rupture of a blood vessel with fatal or serious hemorrhage. When a patient dies from the direct effects of a chronic interstitial nephritis, it is usually due to hemorrhage or cardiac incompensation. The treatment of the terminal stages of this form of nephritis is usually directed toward resting a broken compensation. Edema, when present, is, as a rule, of cardiac origin.

The theories regarding the cause of the hypertension have already been discussed. There is an increasing minority who look upon the hypertension as an effort toward physiological cure. With the impaired ability of the kidney to eliminate solids, the heightened blood pressure causes increased blood flow through the kidney, increased elimination of water and conjointly of solids. The kidney has apparently lost its power to secrete a concentrated urine. The treatment should, therefore, be directed to the underlying cause, rather than directly at the hypertension. With our present knowledge, deficient elimination through the kidney is the apparent cause of the hypertension. Restriction of matter excreted by the kidney and increased elimination through the skin and bowel is a more rational therapy than the use of vasodilator drugs.

Diet.—The proteid diet should be restricted, as in the parenchymatous form, to 80 or at most 100 gms. of proteid daily. If the patient takes 20 gms. daily in the form of vegetable proteid, this leaves 60 to 80 gms. of animal proteid. One liter of milk contains 40 gms., two eggs 12 gms. This would leave 28 gms., providing we are figuring on 100 gms. daily, to be taken in the form of meat proteid. This amount of proteid would be equal to 180 gms. (6 oz.) of raw lean beef. He may take less milk and more meat. The most important factor is that the total proteid taken daily is not in excess of 80 or 100 gms.

The patient may partake freely of the green and root vegetables, cereals, fruits, etc. The only restriction necessary, as far as the kidney is concerned, is in the amount of proteid.

It is quite important that the patient does not become obese. With the heart already overworked, a gain in weight is an added burden. To avoid increased weight, the sugars and fats may be restricted. On account of the danger of overtaxing the heart, severe physical exercise must be avoided, and the patient should depend mainly upon the diet for maintaining the proper weight. Restriction of fluids has been recommended by von Noorden, not on account of the fear of increasing edema, as these patients have good elimination of water, but because he believed excessive fluids increased the work of the heart. This is probably true when very large amounts are taken, but does not hold good if the patient restricts his fluid intake sufficiently to reduce the daily urinary output to 2 liters (2 quarts). Excessive reduction of fluid interferes with the proper elimination through the kidney. Probably the safest rule to follow is to gratify fully the patient's natural desire for water. When confined to water or milk, the patient will rarely take an excessive amount of fluids, unless urged to do so by his physician. Alcohol, if taken, should be limited to very small amounts. This may be permitted in elderly patients who have been accustomed to using it, and where its complete withdrawal may not be well borne. Ginger ale and sharp drinks of this class should be avoided, on account of the danger of renal irritation.

Salt Restriction.—This is less essential in the interstitial type of nephritis than in the other forms, as renal edema is rarely marked. An excessive use of salt causes great thirst; its use may be objected to on this score. The ordinary foods supply sufficient salt, and it is unnecessary to add more. The average patient will soon become accustomed to taking his food with the addition, at most, of very small amounts of salt.

Hypertension.—Hypertension may be treated by increased elimination and by vasodilator drugs. The former method is the more rational. Free catharsis and especially sweating, like all other methods, are transitory. When continued daily for two or three weeks, the pressure throughout the entire twenty-four hours is much reduced, only gradually returning to the previous level after the sweats have been entirely discontinued. By repeating such a course of sweats every three or four months, the patient is much more comfortable and his headaches and dyspnea less annoying. The sweating may be induced by hot wet blankets, hot bricks, or hot air in a special oven or cabinet. The duration of the sweat varies with behavior of the patient. When the respiration and pulse are good twenty minutes is a fair average. When possible, the sweat should be taken with the patient in the horizontal posture, as dizziness and faintness are less likely to occur. A drink of hot lemonade just before entering the bath often facilitates sweating. If the patient feels faint, or the pulse is disturbed, the sweat should be stopped. Discomfort of this sort is most apt to occur during the period of hyperemia of the skin, before perspiration has already begun. The first few sweats are often not as well borne as later ones, and it is advisable to use a lower degree of heat, and shorten the duration in the beginning. When possible, the patient should be kept at rest in bed during the two or three weeks he is taking his sweats. It has been my practice, when possible, to send the patient to the hospital during this period. When he has received daily sweats for ten days, the pressure is sufficiently lowered so that, during the remainder of the stay, sweats every second day are sufficient. During the intervals between these periodical sweats the patient may take one or two sweats per week at his home. Some patients, and this is especially true of those with extremely high blood pressure, are very little affected by the sweat.

Attention should be called to the dangers of sweating. Some patients do not tolerate sweats. After a few days they complain of faintness, palpitation, and weakness. Under these circumstances the treatments should be given less frequently, the duration lessened, and, if these symptoms persist, entirely discontinued. At times the marked reduction of pressure following a sweat may cause alarming symptoms. After a sweat the patient should never be plunged in cold water. Such a precaution may seem unnecessary, yet it is a practice in some institutions after sweating a patient to turn on the cold spray.

In addition to the sweats, the skin should be kept active at all times. Warm underclothing and careful protection from cold. In winter, when possible, these patients should seek a warm climate, as their dyspnea and other cardiac disturbances are more troublesome in cold weather. As a substitute for the sweat, the patient may go to springs where he can take warm baths. These are beneficial, and in addition the patient is improved by the psychic influence of the cure.

Venesection.—This method is in common use for the reduction of high blood pressure. The actual lowering of pressure following its use is slight, not greater than following a sweat. It is also transitory in character as within three or four hours the pressure returns to the previous level. When taking blood pressure before and after bleeding it is well to bear in mind that the excitement due to the prospective bleeding is sufficient to raise the pressure considerably above the normal, and when compared with the pressure after bleeding may give misleading information. In five patients with marked hypertension the blood pressure was taken before and after bleeding, care being taken to avoid the psychic element. From 450 to 600 c. c. were withdrawn. The maximum reduction in pressure was 15 mm. One failed to show any lowering. Where there is threatened or fully developed uremia, or apoplexy, bleeding is indicated, provided the patient is not already anemic. Free catharsis and good elimination through the skin is an excellent substitute for venesection, and is less of a tax on the patient.

Vasodilators.—When the pressure is very high, 200 mm. or more, or when the heart shows evidence of weakening under the increased strain, the vasodilators are indicated. They should not be used in every case of moderately increased blood pressure. There is considerable misconception regarding the actual physiological action of these drugs. They act promptly, but are very transitory in their action. The marked fall in pressure is of short duration. Complete return to the previous level is gradual. As usually administered, three or four times daily, while relieving the heart of considerable labor, they do not maintain the patient's blood pressure at a constant lowered level. Nitroglycerin, sodium nitrite, and erythrol tetranitrate may be used for the reduction of pressure. The first effect on blood pressure when nitroglycerin is placed upon the tongue is noted in about two minutes, the maximum action within six to ten minutes, then gradually rising until after the lapse of fifty minutes it is within ten points or less of the previous level. Complete return to the original level often does not occur under two hours. The usual dose of nitroglycerin is 1-100 grain (0.0006 gm.), best given in the form of 1 per cent. alcoholic solution dropped on the tongue. Many of the tablets on the market are inert. Nitroglycerin has frequently been given in enormous doses—7 grains (0.45 gm.). On the other hand, very small doses, 1-120 grain (0.5 mg.), have caused severe collapse. In a considerable number of patients with high blood pressure (20 per cent. in our series) nitroglycerin does not have any effect on blood pressure in the ordinary doses. These same patients fail to react to considerably larger doses, although the extremely large doses mentioned have never been tried. This failure to reduce pressure is also quite common with sodium nitrite and erythrol tetranitrate. Nitroglycerin not infrequently causes severe headaches, and these may appear after a dose that does not lower pressure. The presence of headache does not necessarily indicate that the pressure has been lowered. Erythrol tetranitrate is placed on the market in the form of ½-grain (30-mg.) tablets. These are chewed and then swallowed. It acts somewhat more slowly than nitroglycerin, the first effect appearing within about four minutes; the maximum within ten minutes. The effect is more prolonged than that of nitroglycerin, persisting even after three or four hours. There is one serious drawback to its use, viz., the frequently severe headaches. These are much more frequent and severe than are noted after nitroglycerin. Sodium nitrite in 2-grain (0.12-gm.) doses is the most satisfactory vasodilator. It acts promptly, rarely causes any headache, and is much more prolonged in its action than nitroglycerin. The above dose may be repeated three or four times daily. Amyl nitrite might be mentioned. It is, however, the least satisfactory of the vasodilators, as its action is so violent that it produces much discomfort and the effect is very transitory. In emergencies it is used on account of promptness of action. A solution of nitroglycerin on the tongue will produce an effect within two minutes, less time than is usually necessary to break the amyl nitrite pearl. It is customary to give these patients iodids in small doses—5 grains (0.3 gm.) three times daily. This dose not affect blood pressure to any appreciable extent, if at all.

Vasodilators are purely palliative. Some have reported permanent lowering of the blood pressure after their continued use. When this occurs, it is probably entirely independent of the drug, as remarkable daily variation in pressure may be noted in untreated cases. At times, for a period of several weeks and without apparent cause, the pressure may average much lower than the normal for that individual. Probably invariably when a patient has hypertension from chronic interstitial nephritis, it is permanent. It may at most only be temporarily modified.

Dyspnea.—On account of the increased work thrown upon the heart by the high blood pressure the patient should carefully avoid overexertion. He may be wisely advised to avoid exercise that makes him dyspneic, as rapid walking, stair climbing or hill climbing, walking against a strong wind, or taking much exercise in the open air in extremely cold weather. Systematic walking with frequent rests, on the other hand, may be beneficial by keeping the heart muscle in better condition. When, in spite of quiet on the part of the patient, dyspnea persists digitalis may be given. With cardiac incompensation pending, digitalis may be of value, notwithstanding the high pressure. The tincture may be used in 10 minims (0.7 c. c.) three times daily, watching carefully for evidence of intoxication, as slowing or irregularity of the pulse. The development of marked cardiac dyspnea in a patient with hypertension is an ominous sign, and usually marks the beginning of the end. Rarely under these conditions can the compensation be completely restored. To relieve the heart of part of its burden, sodium nitrite, 2 grains (0.12 gm.), three times daily, is of value. Free catharsis and sweats often give considerable relief. For the very trying nocturnal dyspnea, from which these patients suffer, next to rest opium is the most satisfactory remedy—the powdered extract in ½ to ¾-grain (0.03-0.045-gm.) doses at bedtime—and usually finally the use of hypodermics of morphia. Under these conditions the ordinary hypnotics, as bromids, chloral, sulphonal, trional, and veronal, as a rule, by depressing the circulation, do more harm than good. The danger of morphinism must be considered, but, as severe nocturnal dyspnea is almost invariably the terminal stage of the trouble, the patient's comfort should be the first consideration. In the earlier stages the Nauheim treatment may be beneficial. Often associated with the dyspnea are attacks of acute pulmonary edema. Hypertension with failing cardiac strength is the most frequent cause of acute non-inflammatory pulmonary edema. Hypodermics of morphin are of value in cutting short the attack. Vasodilators are indicated, but rarely do any good. Atropin, on account of increasing the blood pressure, may be harmful. In suitable subjects venesection is indicated. Oxygen inhalation is also of value. As attacks of acute pulmonary edema are often precipitated by the patient overtaxing in undressing or exposure to chilling, as a cold bath, cold room, or a cold bed, the proper measures should be taken to correct these conditions.

Headache.—Headache is very common in interstitial nephritis. While in some manner probably associated with the high blood pressure, it is seldom relieved by the vasodilators. The coal-tar products most frequently give relief. Phenacetin, 7 grains (0.05 gm.); antipyrin, 7 grains (0.05 gm.), repeated if necessary in four hours, are reasonably safe.

Cerebral Hemorrhage.—Cerebral hemorrhage may be best treated by absolute rest, enforced if necessary by morphin. There is no value in special drug therapy. The skin should be properly cared for by proper cleansing and rubbing with alcohol. The bladder must be watched and catheterization resorted to if necessary. The bowels should not be disturbed for at least forty-eight hours after the hemorrhage, for fear of exciting further bleeding. Later it is important to obtain good elimination through the bowel and skin. After the lapse of two weeks massage and electricity tend to keep the muscles of the paralyzed extremities in better condition, and facilitate use of the part. When the patient recovers sufficiently to move the part he should be put through daily exercises in order to regain accuracy of control of his movements. For the arm movements touching the nose with the fingers, or touching a suspended object, piling up checkers, placing pegs in holes and numerous simple exercises of this character are of the greatest value in acquiring the proper control of the part. Massage is the most efficient measure to prevent contractures.

A cerebral hemorrhage which partially disables the patient tends, perhaps unfortunately, to prolong his life. This enforced rest with freedom from worry and responsibility lowers blood pressure and lessens the work of the heart, and the patient becomes more comfortable than he has been perhaps for years.

Summary.—The chief dangers in chronic interstitial nephritis are those incident to the high blood pressure, viz., cerebral hemorrhage and cardiac weakness. The amount of proteid in the diet should not exceed the minimum body requirement, 80 to 100 gms. It is probably a matter of little importance in what particular form the proteid is taken, whether milk, meat, or eggs. The fats and carbohydrates may be taken freely, unless the patient tends to obesity. Marked restriction of fluids is inadvisable. A sufficient amount should be taken to make the daily output of urine between 1,500 and 2,000 c. c. (1½ to 2 quarts). Marked salt restriction is only necessary in the presence of edema. High blood pressure is best treated by good elimination through the skin and bowel and by freedom from mental strain. When possible, a warm winter climate is advisable. When the pressure is extremely high or cardiac failure impending, the vasodilators are indicated. Of these the sodium nitrite in two-grain doses, three or four times a day, is most satisfactory. To protect the heart, the amount of physical exertion should be carefully regulated. Stair or hill climbing, fast walking, or running should be prohibited. When the cardiac compensation is broken, in addition to the complete rest, digitalis should be given in the form of a good tincture in doses of ten minims (0.7 c. c.) three times daily. For the pulmonary edema, use morphin sulphate hypodermically, ¼ grain (0.015 gm.), bleeding, caffein citrate, oxygen inhalations.

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CHAPTER III

AMYLOID DISEASES OF THE KIDNEY

JOSEPH L. MILLER

The treatment of amyloid diseases of the kidney is purely prophylactic in the treatment of diseased conditions responsible for its development. The two most important causes are chronic suppurative processes and tertiary syphilis. Tuberculosis, with secondary pyogenic infection, is a common cause. Rarely is tubercular infection alone responsible for its development. Chronic suppurative bones and joints, liver abscess, and empyema are common etiological factors. Bull was able to demonstrate amyloid changes in the kidney three weeks after the development of an acute liver abscess. A number of other etiological factors have been held responsible for the development of amyloid diseases. They occur so infrequently and their actual relation to the process is so doubtful that they need not be considered.

The best preventive measures are the prompt and thorough drainage of pus collections and the proper treatment of syphilis. There is no reason to believe that the use of nitric acid, as recommended by Budd, has any specific action. As in many of those cases there are associated amyloid changes in the intestine, with consequent diarrhea, treatment by diet and the remedies used in diarrhea are indicated.

There may be extensive amyloid changes in the kidney with negative urinary findings, and frequently the albumin and casts are due to the development of a parenchymatous nephritis in addition to the amyloid. Uremia probably never develops from a pure amyloid kidney. Edema, when present, is the result of other causes—weak heart, anemia, cachexia, or an associated nephritis. There is never a high blood pressure resulting from pure amyloid degeneration of the kidney. Hypertension indicates that there is an interstitial nephritis in addition to the amyloid. As there are so few renal symptoms, the treatment should be directed at the primary disturbance. The anemia should be treated by diet and iron. When there is evidence of cardiac weakness rest and digitalis are indicated.

Every patient with a chronic disease becomes more or less neurasthenic. He develops a group of symptoms purely nervous in character. In fact, in many mild cases of chronic nephritis, most, or all, of the subjective disturbances may be of this character. It is well to recognize the real nature of these symptoms, and to relieve the patient by the assurance that they are not due to his kidney condition. The importance of psychotherapy is well illustrated in those patients who take up Christian Science. The subjective improvement is very marked; the patient is encouraged and his neurotic symptoms vanish, although the condition of the kidney is unchanged.

The Kidney of Pregnancy.—With the more recent investigation of the toxemia of pregnancy, it has been quite definitely determined that the kidney is less responsible than was formerly claimed. There is a disturbed protein metabolism due to some toxic substance, the exact nature and origin of which have not been determined. The appearance of eclampsia in cases of vesicular mole would indicate that the fetus is not essential to its development. Efforts to obtain a toxic substance from the placenta have given negative results. The toxic substance, presumably a ferment, has an autolytic action on the tissues of the body, more especially the liver. In this organ there is early capillary thrombosis, especially marked in the periphery of the lobule, with destruction of the liver cells and marked tendency to hemorrhage. All the other organs may be involved, usually to a less degree. We are dealing here with a generalized intoxication. When the kidney is markedly affected by this toxin albumin and casts appear in the urine. It is not a localized process in the kidney like that present in acute nephritis. The eclamptic seizure, while resembling uremia, is probably of entirely different origin. The disturbance of partition in the urinary nitrogen, lessening of the urea nitrogen, and increase in ammonia nitrogen may be of assistance in differentiating the toxemia of pregnancy from acute nephritis. Many cases of eclampsia fail to show evidence either clinically or at autopsy of renal involvement. The early recognition of the toxemia is very important. When advanced to the point where convulsions develop the results of treatment are much less favorable. Headache, restlessness, high blood pressure, albuminuria, casts, and increased ammonia nitrogen in the urine may be taken as evidence of the intoxication. The proteid food should be reduced to a minimum, and increase the elimination through the kidney, skin, and bowel. The diet should consist of milk not to exceed a quart, and cereals or cooked fruits. If there is no edema the kidney should be thoroughly flushed by free water drinking. When full blooded, venesection with withdrawal of a pint, followed by transfusion with normal salt solution should be tried. Free sweating is always indicated, and until the symptoms are alleviated the patient may take two good sweats daily. The bowel elimination should be increased by the use of saline cathartics. Foulkrod and others have seen good results following the administration of thyroid extract in grain doses (0.06 gm.) three times daily. When, in spite of these measures, the intoxication increases the uterus should be emptied. Stroganoff has employed the following method of treatment of eclampsia with good results: the patient is isolated in a quiet, darkened room. The convulsions are controlled by the use of morphin hypodermically every two or three hours. In addition chloral hydrate, 10 grains (0.65 gm.) in milk, every hour until five or six doses are given, and if convulsions still tend to appear they must be controlled by chloroform. The patient inhales oxygen freely. Elimination is increased by free water drinking and sweating. As a rule, within twenty-four hours after such treatment is begun labor sets in. When the symptoms do not abate within twenty-four hours, labor must be induced, or the uterus emptied by Cesarean section. Stroganoff lays much stress on keeping the patient quiet, and as free from pain as possible.

Decapsulation of the kidney is of even less value here than in the various forms of nephritis. With a general intoxication, not of renal origin, there is no reason to believe that decapsulation of the kidney will give relief.

CHAPTER IV

PYELITIS

Frank Sherman Meara

Since pyelitis, pyonephritis, pyelonephritis, abscess of the kidney, perinephritis are merely terms of topographical significance, designating the particular locality in which identical processes from identical causes are operative, all of them suppurative inflammations of one or the other or a combination of structures of the kidney, no effort to consider them individually will be made, but the subject will be discussed in its broadest aspect as an infection of the kidney.

The one absolute necessity for the production of a suppurative inflammation of the kidneys is the presence of pyogenic organisms. Unless the numbers are very great or the virulency of the organisms intense, other contributory factors are demanded to occasion the attack.

The organisms most commonly responsible are the bacillus coli communis, the streptococcus pyogenes, and the staphylococcus pyogenes aureus, yet any of the other well-known pyogenic bacteria, such as the staphylococcus pyogenes albus, streptococcus mucosus capsulatus, pneumococcus, bacillus typhosus, paratyphoid bacillus, may be the cause.

The importance of bacillus coli in these infections is becoming constantly more keenly appreciated, and this applies in particular to young children and infants. Colon cystitis, pyelitis, pyelonephritis, and general infection in early life form a chapter in medicine only very recently opened and read.

The routes of invasion of pathogenic organisms are three: (1) by the blood stream, sometimes called descending infection, the route by which most primary infections occur, and usually referred to as hematogenous; (2) through the urinary tract, ascending infection, the common route of secondary infections, and sometimes referred to as urogenous; (3) direct transmission from the colon to the kidney, by way of the lymphatics.

Unless the virulency and numbers of the pathogenic organisms are great, one must assume, and, indeed, careful search is increasingly rewarded with the finding of, local disturbance in the kidney that renders it a locus resistentiæ minoris. In hematogenous infection no doubt the damage done the kidney by the secretion of the toxins of disease has lowered resistance locally and the onset of pyelonephritis or suppurative processes in the parenchyma of the kidney in convalescence so commonly observed favors this view.

It is important from a therapeutic point of view to consider the rôle of constipation and colon inflammation in these infections, because they favor a passage of the colon bacilli into the blood and lymphatics, enhance the virulency of the germ, and, by compression of the ureter, especially the right, by the distended gut, bring about a degree of urinary stasis which contributes powerfully to infection.

The hematogenous infections are commonly bilateral, but by no means always so, for a lesion of some kind existing in only one kidney may determine infection there while the sound kidney escapes.

In ascending or urogenous infection perhaps the most important factors are those that contribute to urinary stasis and abnormal patency of the ureteral orifice, such as to permit a back flow from the bladder.

This interference to the urinary flow in any part of its course, local lesions, and pathogenic microorganisms are the three factors the therapeutist has to keep in mind, with a view to prevention or removal. Renal calculus, inducing damage to kidney substance, pelvis, ureter, or bladder, and so easily obstructing the passages, is always to be early considered; hydronephrosis, tubercular kidney, new growths, stricture, inflammation or blockage by calculus or tumor, or growth in pelvis or ureter along the course of the ureter or at its orifice and the importance of constipation just mentioned, chronic cystitis, "acute cystitis with severe tenesmus and violent expulsive efforts" (Brewer), prostatic enlargement, strictures of the urethra are also to be considered.

The symptomatology of pyelonephritis is often so dominated by the condition to which it is secondary, as, for instance, by the typhoid, grippe, or pneumonia in the descending infection or by the cystitis or calculus in the ascending infection, that the involvement of the kidney may not be suspected unless the examination of the urine has elicited attention, or, again, complications or terminal events, such as uremia or peritonitis from rupture, may be attributed to contracted kidney or appendicitis if the urinary examinations be neglected.

The symptoms referable to the pyelonephritis itself may be divided into those of local and general significance.

The general symptoms are those of fever and toxemia; the local those of pain and urinary disturbance.

The pain is often absent or slight, a soreness or stiffness in the lumbar region, sometimes more marked and interpreted as lumbago. At times the pain is felt in the abdomen, radiating along the ureter and into the thigh, genitals, and perineum, like the pain of renal calculus, or, if on the right side, may be thought that of appendicitis.

The urinary disturbance may be expressed by polyuria, frequency of micturition, or slight pain after voiding, while the urine may attract attention by its cloudy aspect, or much more rarely by the red tinge of a slight hematuria.

The examination determines the presence of pus.

TREATMENT

The management and care of a patient suffering from pyelitis or pyelonephritis are modified by the acuteness or severity of the process. If the attack is acute or the patient suffers from an exacerbation of the process in a chronic infection, or, in other words, if there is a constitutional reaction to the local lesion, evidence of toxemia, the patient should be treated like all cases of acute infection by being put to bed at rest with the best nursing obtainable.

The room chosen should be large, well lighted, well ventilated, free from encumbrances to gather and distribute dust, near a bathroom, and, if possible, leading from a porch or veranda.

The bedstead should be of iron or brass, which can be readily kept clean, half or three-quarters width to facilitate handling, with woven wire springs and a firm mattress. The bed should be neatly made, with the minimum of clothes for the comfort of the patient.

Rest is one of the most important factors in the treatment, and too often taken as a mere phrase.

It economizes the patient's strength, facilitates repair of tissues, lessens the energy demands to be supplied by food, and should demand the same careful attention to detail in its attainment that is given to diet and drugs. Rest requires a well-made bed, a proper toilet, a quiet environment, a rigid exclusion of all causes for worry, anxiety, and useless conversation.

A daily sponge bath for cleanliness, more frequent sponges for fever, care of the mouth, attention to the bowels, fresh air in the room, or removal of the bed to the outside air of the veranda as much as possible in the twenty-four hours are all-important items in the treatment.

When patients are free from fever they are better out of bed, the amount of exercise and work allowed them to depend on their reaction to the same in terms of well-being and weight.

I am firmly convinced that for these patients nothing is of greater value than fresh air, and I warmly advocate the practice of sleeping in the open air at all times of the year, even in the northern climates; of course, with due precaution to the means of protection while in the open.

These patients may take warm baths for purposes of cleanliness and a short, sharp shower bath afterward if the reaction is prompt and satisfactory. Sea bathing introduces the risk of too long exposure, chilling of the damp clothes on coming out, and possibility of undue fatigue, indeed, offers too many risks of lighting up a smoldering process.

Diet.—In acute cases the diet should be bland and nonirritating, but should be sufficient. Certain fundamental principles must be kept in mind in this condition, as in all acute infectious diseases, namely, that the food demands of a febrile patient at rest are not only as great as that of a well man at rest but more in many instances, for pyrexia above 102° F. will ordinarily increase the caloric output some 25 per cent., while the so-called toxic effect on tissue induces even greater katabolic processes. One may take 40 calories per kilo of body weight as a fair working estimate. This would require about 2,800 calories for a man of average weight of 70 kilos, or 154 pounds; or, more roughly, the figure 3,000 may be aimed at for the average man. This food should contain the lower limit of proteid need, which may be taken at 70 to 80 grams, and the proteid, it must be remembered, is the only food with which the kidney is concerned as an eliminative organ, if we exclude water of combustion and salts.

Milk has been shown by practical experience, and approved by theoretical considerations, as the best food for the basis of the dietary. If milk, however, is used exclusively, one of two things happens: too much proteid is ingested to obtain enough calories to meet energy requirements; or, if the proteid is kept within proper limits, the patient is underfed; for one quart of milk contains 640 calories (700 calories to the liter) and 35 grams of proteid. Between four and five quarts would be required for the 3,000 calories and that would furnish between 140 and 175 grams of proteid.

To obviate this difficulty two quarts of milk, furnishing 1,280 calories and 70 grams of proteid, may be used, fortified with milk sugar; while toast, cereals with sugar and cream, make up the balance. An ordinary slice of bread (1½ oz.) furnishes 100 calories; a pat of butter (0.5 oz.) 100; a dish of cereal 100; 2 ounces of cream 100; every ounce of sugar 120. If an ounce of milk sugar were added to each glass of milk in the two quarts nearly 1,000 calories would thus be added.

Other forms of milk, such as buttermilk, or acidified milks, in which the fat is richer than in ordinary buttermilk, may be used, but the caloric value must be appreciated as being lower than the straight milk.

For drinks, water, cereal waters, lemonade, and weak tea or coffee may be given, and sugar added to any of the drinks, of course, has a dietetic significance.

In the chronic conditions a sufficiency of diet is, again, all-important.

Fats and carbohydrates should take a liberal part in it. It should be ordered along much the same line as in chronic nephritis.

It includes meats in moderation, fish, eggs, vegetables, cereals, breadstuffs, simple desserts. It should have variety enough to preserve appetite.

It is less imperative to say what the patient may have than to say what he may not have. The articles to be excluded are all substances which irritate the kidney in the process of elimination or imperil the digestion. They include all condiments, pepper, mustard, etc., radishes, parsley, onions, salted, dried, smoked, potted, deviled, or otherwise preserved meats, rich gravies, and pastries.

Alcohol should be rigidly excluded.

As important as in nephritis is the insistency on simplicity. Three meals a day, sufficient intervals between, say 5 hours, only one hearty meal a day, two or three courses at a meal better than more, and one or two vegetables in a course preferable to a greater number. All food fresh, simply prepared, and thoroughly cooked.

Water should be drunk freely, unless the condition of the circulation or kidney parenchyma forbids.

Local Treatment.—Measures of physical therapy are directed to the kidney region with a view to relieving pain and discomfort and to a possible influence upon the inflammatory process.

Pain is very rarely as intense as in renal colic, though, of course, the pyelonephritis may be the result of the nephrolithiasis, and is often interpreted as a slight lumbago, or the patient may complain of mere stiffness.

For the relief of pain we have recourse to the same measures as in renal colic: hot fomentations, poultices, hot-water bag, or, in some cases, get better results with cold by applying the ice bag (for the technique of these procedures see Nephrolithiasis, Vol. IV, Section I, Chapter VI).

Cups, too, may be applied, preferably dry, though, if there were a great deal of paranephritis, wet cups might afford considerable relief.

For cups one may use small medicine glasses, egg cups, wine glasses, or any thin glass with a wide bottom.

The skin over the kidney region should be cleansed, and as many cups as the part will accommodate without interference applied.

In severer grades of pain the actual cautery may be lightly applied to the surface of the skin.

In some cases the pain may be severe enough to demand the administration of a drug, and this should be small amounts of coal-tar, such as phenacetin, gr. v (gm. 0.3), or acetanilid, gr. iii (gm. 0.2), every half hour or hour for 3 doses; or, if this fails, morphin as a suppository, gr. 1/4 to gr. 1/3 (gm. 0.018-0.020), or hypodermically, in doses of gr. 1/8 to gr. ¼ (gm. 0.009-0.018), the method and dose depending on the severity of the pain.

Bowels.—The condition of the bowels should be a matter of concern, for neglect may entail not only the usual results of malaise, headache, impaired appetite, load the products of fermentation and putrefaction absorbed from the bowel upon the blood, but constipation and its results enhance the virulency of the Bacillus coli, so often the microorganism concerned, facilitate its passage into the lymph or blood vessels, while the distended gut by pressure on the ureter encourages stasis of its contents and forwards the process of infection. In the chronic cases the diet should contain enough coarse material, cellulose or seeds of fruits, to relieve constipation, or mild cathartics, such as compound licorice powder or saline waters, may be used. In the acuter cases salts or enemata.

Diuretics and Urinary Antiseptics.—Whenever a deleterious substance enters the body or appears in the body two mechanisms for protection are immediately set at work: first, one to render innocuous or destroy the deleterious substance, either by chemical combination or the elaboration of immune bodies; and, second, by elimination. The three great eliminating organs or emunctories are, then, or ought to be, objects of especial solicitude in any infection, and their functions jealously preserved or encouraged. We have emphasized the care of the skin and bowel, but in the disease under consideration the third great emunctory is itself the site of morbid processes, yet must perform its share in the work of elimination. The importance of eliminating from the diet all irritating substances that might further burden the kidney has been touched upon, but it is equally important to remember to exercise the same precaution in the choice of drugs and exclude any that might cause the inflammation to flare up, remembering that even drugs applied to the skin may affect the kidney, as in the application of cantharides.

The best diuretic is water, and its ingestion should be encouraged in the shape of plain water or water flavored with fruit juices or a mild diuretic like Imperial Drink (cream of tartar, ℥ss to Oiii boiling water, with sugar and lemon peel to the taste, to be taken ad libitum); or the water in the shape of a physiological salt solution (roughly, a teaspoonful of salt to a pint) may be given by the bowel, as an enteroclysis (see Nephrolithiasis, Vol. IV, Section I, Chapter VI), a slow irrigation, or as a Murphy drip, using the term as a slow continuous irrigation.

My own apparatus consists of a thermos bottle, with a perforation in the top, through which a tube connects with the irrigation tube, while a perforation in the bottom of the inverted bottle admits the air. This apparatus keeps the temperature even with the least trouble. The tube from this bottle is pinched by a cock in such a way as to regulate the flow from the bottle.

One may use any kind of a container. The rectal tube may be hard rubber, glass, or soft rubber, but, in case of the former, the shape should be such as to avoid continuous pressure against the wall of the rectum.

If the tube is not regulated by a cock the container should be raised only about 6 inches above the level of the patient's buttocks, that is, at such a height as to give a hydrostatic pressure sufficient to distend the bowel gently, but not excite peristalsis; if the inflow is regulated by the cock the height of the container is immaterial.

Whether the absorption is facilitated by the distended bowel I am not prepared to say.

The temperature of the physiological salt solution used is 100° F.

The time varies from 2 to 3 hours to 24 hours in the day, depending on results and the manner in which the patient bears it.

In severe toxemias, or in threatened anuria, a hypodermoclysis or an infusion may be indicated.

The effect of the diuresis is a flushing out of the pelvis of the kidney, ureters, and bladder, a kind of natural irrigation.

The urine may be acid or alkaline. In the case of colon bacillus or typhoid pyelitis, the urine is usually acid, in the streptococcic and other forms of infection, which are secondary to an infection lower down, it is usually alkaline.

Changing the reaction of the urine seems to afford a less suitable medium to the bacterium concerned, so, in case of an acid urine, alkaline salts, such as bicarbonate of soda, citrate of potash, or acetate of potash, or acid potassium tartrate, i. e., cream of tartar, may be used in doses of gr. xv to gr. xxx (gm. 1-2), singly or in combination, every two hours until the urine is alkaline, and then the dose reduced in amount or frequency until only enough is given to keep the urine alkaline.

If, on the other hand, the urine is alkaline, benzoic acid, preferably as one of its salts, sodium or ammonium, or salicylates of sodium or aspirin, in doses of gr. x to gr. xx (gm. 0.6-1.3) or more every four hours, is indicated until the object is accomplished and then only enough to keep the urine alkaline.

Benzoic acid, eliminated as hippuric acid, and salicylic acid, eliminated as salicyluric acid, are antiseptic in their qualities, but the one drug which at present seems the most efficacious as an antiseptic is hexamethylenamin, chemically, hexamethylenetetramin, which, in its own form or that of its derivatives, is known by several trade names, urotropin, helmitol, formin, etc., a regrettable multiplicity of nomenclature, that has led the careless on occasion to change from one to the other on the supposition of a difference in their chemical constitution and pharmacology.

Here we shall deal only with its older and better-known usage, as a urinary antiseptic, but it is well in passing to call attention to later investigations, which show its elimination into the bile, the cerebrospinal, the pleural and synovial fluids, as indicating possible therapy in these respective fields.

The activity of the drug seems to be dependent on the liberation of formaldehyd, which is said to occur in the course of its elimination by the urinary tract. It is maintained by some authorities that this liberation of formaldehyd occurs only in an acid medium, and, guided by this belief, some clinicians insist on the simultaneous administration of benzoic acid or its salts. I am not convinced that this contention is surely established, but call attention to it as worthy of trial before urotropin medication is abandoned as useless in a given case. The dose should be gr. xx to gr. xxx a day, gr. v to gr. vii ss (gm. 1.3 to 2.0-0.3 to 0.5) in water 4 times a day.

The drug may be administered for a long time without evidence of harm, though occasionally in large doses, or in patients peculiarly susceptible, hematuria may occur or gastric unease, bladder distress, and abdominal pain, and diarrhea. These symptoms subside promptly on withdrawal of the drug.

All bacteria are not equally susceptible to its influence, the typhoid bacillus and the bacillus coli seem the most so.

Local Treatment.—When the pelvis alone is affected, injection of silver nitrate through the ureteral catheter has been reported favorably to the amounts of ℥i of 1 per cent. solution.

Vaccine Therapy.—Studies in immunology have brought to the practitioner hope in abundance and results in some measure.

A desperate condition will know no denial, so the physician with a measure at his command that has so many theoretical considerations in its favor, even though the statistics of success are yet scarce, will, providing he feels that no danger ensues from the procedure itself, hardly deprive the patient of the chance it offers.

In pyelonephritis there are two conditions which may prevail that would invite to vaccination: first, a persistency of the local process, a pyelitis or pyelonephritis that has resisted the older measures and is becoming chronic; and, second, the septicemia that may result. The former alone offers much promise to the effort; in the latter condition it is a *dernier ressort.*

As yet but few cases have been reported, but the statistics read encouragingly. Stoner (3), in a summary of vaccine work done up to 1911, reports 14 cases of pyelitis treated by this method, with 8 cases cured and 3 improved.

All of these cases were due to the bacillus coli, one mixed with tuberculosis.

A case of my own, due to streptococcus mucosus capsulatus, mixed with pyocyaneus, in whom a nephrectomy was done three years before his death, owed, in my opinion, a freedom of constitutional symptoms, a gain in weight to the maximum of his life, and strength to conduct a large business with heavy responsibilities during nearly all the interval to the use of autogenous vaccines, but in his case, as has been repeatedly observed, the urine made but little improvement, in spite of the great improvement in well-being. In all cases, however, as in this, open-air sleeping, careful hygiene, and diet must be observed.

Whoever undertakes this mode of therapy should acquaint himself with both the theory and the technique. It is not to be undertaken lightly nor carelessly, and general instructions are no more than points of the compass; the path to be followed is determined by a conscientious study of the individual patient and his reaction to each injection.

Autogenous vaccines should always be used, if possible, both because the organism concerned is certainly established and because the antibodies produced are so specific that even other strains of the same organism do not give as good results.

Stock vaccines may be used where the nature of the organism is determined, and the delay in the preparation of the vaccines may prejudice the patient's condition, or where a culture may not be obtained and the clinical features of the case make the diagnosis fairly certain, as, for example, that over 50 per cent. of the cases of pyelitis are due to the bacillus coli and that the urine is characteristically acid in these cases might warrant the use of stock colon vaccines.

For further details the reader is referred to the chapter on Vaccine Therapy, Vol. I.

The number of injections vary with the response from three or four to a dozen or more.

It might at first seem irrational to expect a body already overwhelmed with poisons to react favorably to the imposition of more, but the formation of antibodies would seem to be a local reaction at the site of the inoculation and occurs when the tissues at the site of the original infection can no longer respond; and these antibodies escaping into the circulating fluids effect in their own particular way a decrease of the toxins.

Operation.—The operative procedures one must have recourse to in a certain number of these cases can be referred to here only in the briefest manner; for detail reference must be made to a surgical treatise.

If an uncomplicated perinephritic abscess exists, incision and drainage are indicated; if a pyelonephrosis without implication of parenchyma, a pyelotomy; but with involvement of the kidney substance itself the question of operation resolves itself into a choice between nephrotomy and nephrectomy.

If nephrectomy is to be done it is imperative to know the functional ability and structural integrity of the remaining kidney; and, as the decision between the two procedures may not be settled until the kidney is exposed in the course of the operation, this functional test should be made in each instance. The three constituents of the urine to be determined are water, salts, and nitrogen.

Although in certain forms of nephritis sodium chlorid is discriminated against particularly, by the kidney (the determination is not difficult), still it is the first and last functions that concern us here.

Catheterization of the ureters readily determines the output in a given time of one and the other kidney in terms of water and nitrogen, and, moreover, affords a study of the exudate from each kidney.

Allowance must be made for certain variations in the functions of the two kidneys at any one time, even in the normal subject; but the studies of Baringer and Baringer (1) and those of Albarran cited by them show that, in the normal subject, this variation is not great.

Innumerable methods have been employed to determine the question of functional integrity, physical methods such as cryoscopy, electrical conductivity of the urine, the eliminations of drugs: potassium iodid, the salicylates, phloridzin, dyes—such as methylene blue, rosanilin, etc. One of the most attractive and promising of these tests is that with phenolsulphonephthalein, which is described in detail with a review of other methods recently by Rowntree and Geraghty.

Complete destruction of a kidney or serious involvement of the structure, multiple abscesses, would indicate immediate nephrectomy. With a lesser degree of impairment of one kidney and integrity of the other it is a nice question, determined more or less by the localization of the process, whether it is better to do a nephrotomy and drain, hoping to retain a goodly portion of the damaged kidney; and, failing of prompt healing, do a nephrectomy later; or, whether it is better to do a nephrectomy at once, fearing that the retention of a damaged kidney may imperil the other before nephrotomy has demonstrated its efficiency; or the secondary nephrectomy is done. I think, in the light of the compensatory growth of the remaining kidney, and its capacity for meeting the excretory needs of the body, the tendency is toward taking no chances of imperiling the sound kidney and favoring immediate nephrectomy.

If both kidneys are involved the question of nephrotomy or nephrectomy must depend on the relative involvement of the two kidneys. A suppurative process may still be compatible with a high degree of functional capacity; and the excision of a kidney that has no function and simply pours its bacteria and toxins on the relatively good kidney is certainly indicated. On the other hand, if both kidneys are badly impaired, the excision of one will precipitate a final issue, to which the operation itself may be the determining factor.

COMPLICATIONS

The pyelonephritis may be associated with, indeed due to, tuberculosis of the kidney (see Tubercular Kidney) or renal calculus (see Nephrolithiasis), and may introduce the problems of these diseases.

Cystitis.—Cystitis may be due to a pyelonephritis of hematogenous origin, but is more commonly the condition to which the pyelonephritis is secondary by ascending infection. The treatment of the cystitis, then, is a most serious consideration, involving the removal of the exciting cause, such as stone, enlarged prostate, changing the reaction of the urine, use of urotropin, bladder irrigations, and the use of vaccines. In Stoner's article, referred to, 83 cases so treated are cited, with 47 cured and 26 improved.

Retention of Urine.—This may occur, most commonly due to hypertrophied prostate, and, as has been said earlier in this article, powerfully favors the advance of infection. Prostatectomy or a catheter life are the alternatives.

Suppression.—Suppression is an ominous symptom, and demands all the efforts directed at this condition under other circumstances. To suppression will probably succeed uremia, in the combating of which the measures usually pursued (see Nephritis) are attempted as a forlorn hope. The infection from being local may become general and a true septicemia follow. Here the measures applied in any acute infection obtain: fresh air, sufficiency of easily digestible food, cold water to control temperature, if excessive; heat to the skin and internally when chills occur. The use of vaccines, in the smaller doses more frequently administered. When such a condition prevails our efforts are to make the patient comfortable, in which we may be successful, to combat the infection by whatsoever measure holds out any promise, but at the present moment unfortunately with very little likelihood of success.

Peritonitis.—Peritonitis is a rare accident due to rupture of an abscess into the peritoneum, and must be dealt with surgically.

Cardiac Hypertrophy and Hypertension.—This is an expression of interstitial changes in the kidney, and is to be considered in the same light as when occurring in chronic interstitial nephritis—support for the heart when the compensation breaks, and study of the hypertension to determine at what point this increased blood pressure constitutes an optimum for the individual patient.

Gastrointestinal Symptoms.—These are sometimes a source of great discomfort to the patient, and are often the manifestation of uremia. Gastric sedatives, bismuth, soda, cerium oxalate, gastric lavage, cracked ice, cocain, or even morphin, in the worst cases; local measures, heat to epigastrium, or cold, or counterirritation are all to be tried. Diarrhea must be controlled, if excessive, by bismuth or opium, but it must not be forgotten that uremic diarrheas are probably of purposeful significance in the elimination of poisons.

PROPHYLAXIS

Prophylactic treatment begins with all those rules of hygiene, diet, and exercise that make for a healthy body.

Next, it takes cognizance of possible causes of suppuration of the kidney and endeavors to remove them or render them less likely to become operative.

Recognizing the acute infectious diseases as possibly excitant of the process by hematogenous infection, a rational treatment of the disease concerned, an abundance of water, care of the other emunctories, and especial attention to local lesions in the course of the disease, such as tonsillitis, otitis, adenitis, or abscesses, lessen the likelihood of pyelonephritis. The administration of urotropin in the latter days of a typhoid fever is a preventive measure of importance. Considering the possible sources of an ascending infection, suppurative processes or inflammations or mechanical impediments in the urinary tract should receive attention, such as gonorrheal urethritis, cystitis from any cause, hypertrophied prostate, pelvic masses that might be obstructive, a bacilluria without obvious signs of inflammation, movable kidney with Dietl's crises.

Instructions should be given to mother and nurse in the light of the frequent infection of female infants and children to wipe excretions away from rather than toward the vulva, and observe strict cleanliness of the parts and clothes.

Appreciating the road direct by the lymphatics, and that infection through the blood may come from the same source, the bowel should receive attention, constipation be avoided, and bacilluria, usually from bacillus coli, during intestinal disease, be early detected and treated by urotropin, abundance of water, or by alkalies.

Finally, attention must be given to diseases of the kidney itself, to which pyelitis and pyelonephritis are common, to eradicate them as far as possible, namely, nephrolithiasis, tubercular kidney, and hydronephrosis.

Surgical cleanliness must be observed on the occasion of every necessary instrumentation of the genitourinary tract.

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CHAPTER V

HYDRONEPHROSIS

Frank Sherman Meara

Hydronephrosis may be either congenital or acquired; it is with the latter that we are especially concerned in this chapter.

Acquired hydronephrosis is the result of an obstruction, partial or complete, to the flow of urine anywhere along the course of the urinary passages. It is more likely to be bilateral, if the cause is operative in the bladder, prostate, or urethra. Pelvic tumors, growths, or peritonitis may involve one or both ureters, depending on the extent.

A movable kidney, by causing a kinking of the ureter, is another potent cause. Pelvic tumors, inflammation, and movable kidney make the condition more frequent in women than in men.

Perhaps the most common cause is renal calculus, which causes stenosis of the ureter, on the one hand by impaction and on the other by cicatricial contraction secondary to trauma and ulceration caused by the passage of the stone. Stone and movable kidney are much more likely to induce unilateral lesion.

Four results are likely to ensue upon the development of a hydronephrosis, each of which has both local and remote results:

First, the tumor itself, which, from its size, interferes with the relationship of the surrounding organs, and in some measure with their function.

Second, the huge collection of water in the pelvis induces atrophic changes in the parenchyma of the kidney, even to the point of complete destruction, thus depriving the body of the function of that organ and throwing all the work on the other kidney.

Thirdly, if Castaigne and Rathery were correct in their insistence, there is always a lesion of the opposite kidney, which, from their clinical and pathological observations, and from their animal experimentations, they attribute to the toxic effects of the products of cell destruction in the nephrotic kidney, and they assert that the serum of an animal on whom a ligature of the ureter has been practiced acquires nephrotoxic properties. Thus a chronic nephritis is induced in the opposite kidney, with terminal uremia.

Fourth, these kidneys are especially prone to infection, and a pyelonephrosis or pyonephrosis set up with consequent general toxemia or septicemia.

Hydronephrosis may be permanent or intermittent, and it is the latter type that affords a very interesting clinical picture. It is almost invariably unilateral, and is due to the kinking of the ureter of a movable kidney or to an impacted calculus, the occlusion by which is overcome by the collecting urine.

The characteristic feature of intermittent hydronephrosis is the formation of a huge tumor, which suddenly disappears with the simultaneous passage of a very large amount of urine. With the accumulation there may be pain, at times excruciating, and relief on disappearance of the mass.

In an interesting case I had for some time under observation the collection would be accompanied by severe pains, like renal colic, and gradually disappear, the huge collection remaining, at times, for weeks, then, without warning, emptying itself, either to remain empty for a long period or to reaccumulate at once. It would seem probable that the degree of pain would depend somewhat upon the rapidity of accumulation and be the more severe in a kidney which had not yet undergone much change.

TREATMENT

Bilateral congenital hydronephrosis is incompatible with life. Unilateral congenital hydronephrosis will compel surgical intervention.

In the case of acquired hydronephrosis the question to be decided is whether operation is or is not indicated.

Patients have gone on for years with no apparent ill effects from the condition until the parenchyma of the kidney has been destroyed, and this knowledge, no doubt, often persuades to conservative treatment, and, in the intermittent cases, a failure to refill and a spontaneous cure has been known to occur, but the danger of infection ascending or descending is always present, and the more remote possibility of rupture from traumatism.

The rational procedure is to set about to determine the site and nature of the obstruction, its degree, the integrity of the affected kidney, and the presence or absence of any lesion of the other. This involves exploration of the urethra, cystoscopy to determine obstructive conditions in the bladder, catheterization of ureters, to determine the degree of stenosis, its site, perhaps its character, as the use of wax tip in searching for stone, the collection of urine from either side to determine rate of flow and character of urine, thus discovering the amount of functional impairment of the affected kidney, and evidences of nephritis in the other. X-ray plates should be secured to detect a possible stone, and thorough abdominal, rectal, or vaginal examinations made.

> I am convinced that, with the perfection of technique of to-day in this field of surgery, an operation is always indicated, which, exploratory at first, proceeds on the basis of its findings.

If a stone is found, and the previous examination of the urine from that side or the appearance of the kidney warrants the assumption of a functioning organ, the stone is to be removed and the kidney saved; if a movable kidney is discovered and a restoration to its normal position promises to relieve the condition, the kidney being still functioning, this operation is indicated. Relief of constriction or plastic work on the ureter may be indicated.

If the kidney has been practically destroyed, or the obstruction cannot be removed, or the kidney is infected, it remains but a menace to the other kidney and the life of the patient, and nephrectomy should be done, or nephrotomy, followed by a secondary nephrectomy, depending on the findings at the operation.

For the attacks of pain, the same local and drug measures may be used as in renal colic (see Nephrolithiasis).

In some instances the patients contrive to find a position that effects a release of the accumulation, and at times manipulation of the tumor mass, in an effort to elevate it or gently massage it, does the same; but extreme caution must be used in such efforts.

Aspiration has been undertaken, but seems to me justifiable only in cases of extreme pain not controlled by other means, and where operation cannot be done. It relieves the distention and affords temporary relief.

The site usually advised for paracentesis is on the left side, just anterior to the last intercostal space; but on the right side, owing to the interposition of the liver at this site, lower down, halfway between the last rib and the iliac crest and two inches posterior to the anterior superior spine of the ilium.

After the attack of pain, if operation has been refused, effort may be made by seeking various positions or by the use of binders and belts to effect an unkinking of the ureter.

If infection has occurred and the hydronephrosis converted into a pyonephrosis, operation is doubly imperative; if refused or not feasible the same line of treatment is carried out as suggested under similar circumstances when treating pyelitis.

Cystitis is to be treated, as under other circumstances, by local applications, drugs, and irrigations.

The other kidney may not go scot free, and, as has been said, it is maintained by good authority that the presence of the hydronephrotic kidney, not its absence, imperils the other kidney, and a sclerotic condition may ensue, with uremia as the result. Our task is to appreciate the involvement of the second kidney before this accident occurs, and take such measures to reduce its task by dietetic and hygienic precautions as in any case of chronic nephritis. The development of uremia entails the usual procedures for this condition (see Uremia), but with the knowledge that the fight is that of a forlorn hope and the appreciation that the best we can do is to lessen suffering by controlling the gastric distress and preventing the onset of convulsive seizures, more distressing to family and friends than to the patient at this stage.

CHAPTER VI

NEPHROLITHIASIS

Frank Sherman Meara

Nephrolithiasis is a condition which, in one or more of its manifestations, must be met repeatedly by every general practitioner of medicine; a condition in the consideration and treatment of which the internist, the surgeon, the genitourinary specialist, and the roentgenologist are called upon for their best endeavor.

The gravity of the affection depends very largely on the presence or absence of infection. As long as the patient is free from infection his symptoms are referable only to the mechanical effects of the stone, the pain incident upon its movement through the tract, the disturbance of function induced by the irritation, and the obstruction ensuing upon its impaction; but, when infection obtains, the case takes on a new and more serious aspect. The trauma, stagnation, and interference with function attributable to the mechanical effects of the calculus render the field fertile to the seeds of infection, which may arrive at the point of least resistance either from below, by way of the genitourinary tract, the so-called ascending infection, or from above, by way of the renal circulation, the so-called descending or hematogenous infection.

While in the vast majority the stone is primary and the infection secondary, there is still a group of cases in which a previous infection of the urinary passages has furnished the conditions under which stone formation may occur.

While it is possible for calculi to be present in the form of sand or of the coarser gravel or even at times as a considerable stone, and give rise to no symptoms whatsoever; and, while such symptoms as are present may be of a lesser character, giving rise to discomfort and dull backache, which may be attributed to lumbago or neuralgia; or, finally, while the infection, as a pyelitis, a cystitis, or a pyelonephritis, may dominate the picture, nevertheless our earliest introduction to the condition and our most common experience with it are, as a rule, on the occasion of one of those violent crises of pain termed a renal colic.

The pain may be of any grade of severity; as a rule, very decided, often agonizing, beginning in the lumbar region and radiating in a characteristic way around the side of the abdomen to the groin, into the scrotum and testicle, or into the labia majora in the female, often into the penis, down the inner side of the thigh, and at times into the rectum and perineum. Nausea and vomiting are common accompaniments of the pain, as they are of severe attacks of hepatic colic. The pulse becomes rapid, the patient is prostrated, or may go into collapse. Not infrequently a chill occurs and fever follows, and this, too, in cases not recognized as septic. Where infection has taken place the chills and fever may be so severe and frequent as to give rise to the diagnosis of malarial paroxysms. Frequency of micturition may or may not take place, and, according to Young, depends on the position of the stone, those which are low down in the ureter, intramural, or projecting into the bladder, exciting desire to urinate.

Sometimes suppression follows and may continue for hours or days, due more often to cessation of function from irritation than from blocking.

The attack may be very brief or last for hours, leaving a dull ache as an aftermath, while the urine voided shows, from the red and white cells it contains, the trauma suffered by the passages.

It seems advisable to take up the treatment much as the demands are made on us in practice rather than in the more formal manner of a treatise.

TREATMENT OF RENAL COLIC

Pain, and pain alone, is the consideration of the moment. The demand for relief is imperative. All of the immediate symptoms are due to the pain and disappear with the pain.

With the first premonition of an attack or in attacks of lesser severity relief may be afforded by the use of heat in the shape of a hot bath. It is one of the procedures the patient himself may initiate, awaiting the advent of the physician. It is possible that the attack may be aborted or greatly ameliorated by this means.

The patient enters a tub containing enough water to submerge the trunk and limbs. The temperature of the water on entering should be about 100° F. and gradually increased to 110° F. to 115° F., depending upon the patient's sensitiveness to heat. Cold applications are made to the head during the bath. The bath is continued for 5 to 15 minutes, being interrupted on the occasion of any feeling of faintness. The effects may well be continued by rolling the patient in two or three dry blankets which are made to inclose the body snugly, while cold is still applied to the head. In the pack the patient often sweats profusely. When taken from the bath he should be rubbed dry, given an alcohol rub, but the body still kept warm.

The rationale of this procedure depends on the effect of warmth on the action of muscle tissue.

Such experimental data as we have at hand point to a diminution of tone of both striated and smooth muscle under these conditions. Such an effect would mean a relaxation of spasm, and thus experiment has confirmed experience, for the relaxing effect of the warm bath in renal colic has long been accepted by practitioners of medicine.

Local Application of Heat.—When, from the circumstances of the environment, a hot bath is not obtainable, or from the severity of the pain, or the weakness of the patient, it is not feasible, heat applied locally may afford some measure of relief.

Hot fomentations: The most rapid and efficacious method of applying heat is in the shape of cloths wrung out of hot water. Two or three pieces of thick flannel cut to the size of the site of application are saturated with boiling water, as they lie in a crash towel or other container, the ends of the towel being twisted in opposite directions to squeeze out the excess of water from the flannels it contains. Sticks attached to the ends of the towel facilitate the process. The site of application is over the lumbar region of the affected side or over the site of greatest pain. The skin is smeared with oil or vaselin, the degree of heat of the compresses tested to avoid a burn, the cloths then laid on smoothly, and a dry flannel placed over them.

The fomentation is left on for 5 or 10 minutes, then another applied until three or four have been done. If the procedure has been efficacious for the time, it may be repeated on a recurrence or aggravation of the pain.

Poultice: While the poultice is not so readily prepared as the fomentation, it has the advantage of affording heat for a longer time and sparing the patient the disturbance of repeated applications. Perhaps the best material for the poultice is flaxseed meal.

The hot-water bag or bottle: In no form can heat be more readily applied; but the application cannot be so intimate throughout the area to be considered as with other methods. Care must be taken that there is no drop or leak, and sufficient flannel intervene between bag and skin to regulate the temperature. The bag should not be more than half full, the air expressed, by squeezing the water to the nozzle level, and the cap screwed firmly on.

In this way the bag becomes more pliable and makes more easy its application and retention.

Hot douches: While affording another means of applying heat locally, it is rarely attainable except in institutions, and not applicable in the severe cases.

These measures, like other measures of physical therapy, are our best heritage from empiricism, and the belief in their efficacy has been fostered much more constantly by the laity than by the profession. Just at present, to my mind fortunately, physical therapy is enjoying a renaissance. Much work has been done to analyze the effects of these physical agents, yet it is at the best but pioneer work.

As to the rationale of the application of heat locally in the condition under discussion, I cannot do better than quote Matthes' summation of our knowledge of such effects, namely, "One can actually influence the distribution of blood hydrotherapeutically (1) through the local application of heat or cold, which operates directly at the site of application, upon the vessels or reflexly; or (2) through the reaction upon other vascular provinces exerted hemodynamically or reflexly. We can further assert that we are in a condition to render the skin richer or poorer in blood and that we can in this way exert a considerable influence on the circulation of the internal organs. Unfortunately this influence on the circulation of the internal organs is very incompletely known in its details" (2).

It is possible, then, that a relief of congestion or inflammation induced by the trauma may be afforded in this manner, and that the relaxing effect upon the ureteral structures occurs from the local application of heat as it is believed ensues when the application is general, as in the bath.

Cold.—Some cases are said to find more relief from the application of cold than heat, but I think they are relatively rare. The best mode of application is the ice bag. I prefer the circular one. Use small pieces of ice, enough to cover the bottom, water enough to cover the ice, and express the air to render the bag supple and easy of application. Put gauze or muslin between bag and skin.

Enteroclysis.—Warm water in the bowel may determine some relief of pain. It is best used in the shape of an irrigation. The temperature of the water should be 100° F. to 105° F., the quantity 1 to 3 pints, to which has been added common salt in the proportion of ʒi to the pint (physiological salt solution). This is introduced into a fountain syringe or douche bag, held 3 to 5 feet above the level of the bed. A two-way tube should be used, the most easily improvised being two catheters, one of a slightly smaller caliber than the other and protruding slightly beyond the other, the smaller for the introduction of water and the larger for its discharge and that of the small solid particles that may accompany it. These may be kept together by silk ligatures, or may be sewed together. The tubes should be well oiled before introducing, and no force used in the procedure. If obstruction is met, wait until any spasm provoked has ceased before continuing. Allow a little water to flow, thus distending the bowel ahead of the tube and facilitating its passage. Pass the tubes in 6 to 9 inches. A Kemp tube, especially devised for the process, may be used.

A relaxing effect on the spasm of the ureter by the water in the contiguous structure probably exerted reflexly may be assumed. The diuresis provoked by the procedure may also be helpful in dislodging the calculus.

Warm Drinks.—Warm milk, barley water, Vichy, or lemonade may be given with benefit. Probably a relaxing effect is induced by warm water taken internally, as it is when locally applied; at least, fall in blood pressure attributable to lowered vascular tone has been observed after the ingestion of warm drinks.

Posture.—There are certain postures in which the patient may find the greatest relief. He will most certainly seek such, and the position should be respected as much as possible by the physician in his procedures. Occasionally inversion of the patient is efficacious.

The above measures are applicable only in the attacks of lesser severity, or when the severity of the attack has been lessened by other measures. In many cases, perhaps in most cases, of renal colic the demand for relief is so urgent that immediate recourse must be had to drugs.

Drugs.—There is no hesitancy in the choice of drugs under these circumstances. Morphin stands preëminent. It must be given hypodermically, down into a muscle for a more rapid effect, and it must be given in sufficient quantity. Not merely age and weight, but the pathological condition treated determines the dosage of morphin, and severe pain requires large doses. Indeed, in the cruelly punishing attacks of colic, amounts of morphin that trespass closely on the danger line under other circumstances may neither relieve nor manifest the usual toxic effects. In the adult, if the colic demands morphin, gr. 1/4 should be the beginning dose, and, if some result is not seen in a quarter of an hour, another dose of gr. 1/8 may be given, which, in turn, may be repeated in the same length of time.

Lesser doses but delay the effect, the patient is nagged by the frequency of administration, and the physician left uncertain as to what his next dosage should be when several small ones have already been administered. The same unsatisfactory train of events ensues if attempts are made in the severe cases to begin administration by mouth or rectum, and, as our doses are usually larger when given in this manner, we are left uncertain, when we feel compelled to give a hypodermic, as to just how much has already been absorbed and what our hypodermic dose shall be.

CHLOROFORM.—Another drug that occupies in this therapy a place second only to morphin, and, in exceptional cases, first, is chloroform.

In the most violent attacks morphin may not afford relief in doses that the practitioner is willing to give. I have given, in a case of hepatic colic, gr. 3/4 of morphin in a very short space of time without effect. I did not care to go beyond that. In these cases a few whiffs of chloroform will afford relief, but often not before the patient is mildly anesthetized. When the intensity of the spasm has thus been broken up, the morphin previously administered becomes operative and is efficient in preventing the recurrence of the attack.

In these very severe cases it is often well to give the chloroform at once, and then, with any premonition of recurrence, give morphin, gr. 1/6.

The chloroform should not be pushed or continued for any length of time, but enough given to relieve the spasm.

Its effect may be permanent, owing to the discharge of the calculus during the period of relaxation or to the relief of the pressure behind the obstruction, the relaxation permitting of the passage of urine by the impacted calculus; or its effects may be evanescent owing to a recurrence of the original conditions giving rise to the attack.

In these latter cases the addition of the morphin is needed.

The usually accepted view of the cause of the colic is the spasm set up by the irritation induced by the calculus; Bevan, however, is confident that the passage of the stone through the ureter itself does not cause any pain at all, but that "the cause of a renal colic is a greatly increased intrarenal tension due to a plugging of the ureter" (1); and, while the former view is the simpler, still the rapid disappearance of the attack with relief over long intervals, though the stone is shown by the X-ray to remain in situ, the passage of stones of considerable size and roughness without pain, and the attacks of colic induced by blood clots or other soft substances add weight to Bevan's view.

Another indication for chloroform in preference to morphin is in those cases who have a decided idiosyncrasy for morphin.

Finally, one feels warranted in giving patients who are prone to such attacks and likely to be taken when remote from assistance the advice given by Lauder Brunton to patients suffering from angina pectoris, namely, to place a sponge in a small bottle with a wide neck, and, on the occasion of an attack, wet this sponge in chloroform and inhale. As the patient drowses off, the bottle will fall from his hand, and no harm from overdose can occur.

Atropin.—Because of the striking effect of this drug on all unstriated muscle substance, whose function it diminishes, it is used when these structures are in a condition of spasm. The best way to administer atropin is hypodermically, and a sufficiency used to effect the purpose: gr. 1-100, gr. 1-75, or gr. 1-50 (gm. 0.0006-0.0012).

It may be used in combination with morphin, gr. 1-150 to gr. 1-75, thus bringing the sedative effect of the two drugs to bear at once. Each tends to bring relief, though the modus operandi is not identical.

Another advantage of the combination of the two drugs lies in the fact that the nausea and vomiting, which in many patients are the inevitable sequelæ of morphin administration, are greatly ameliorated or quite prevented by the atropin.

Here, again, one must be assured that the patient has never shown marked idiosyncrasies for the members of the belladonna group.

Chloral.—When the pain is less severe, or has in a measure been controlled, chloral, on account of its sedative effect on all nerve structures, may break the reflex between sensory irritation and muscular response in the ureter and thus bring relief. It may be given by the mouth in doses of gr. xv-xx (gm. 1.0-1.3), or by rectum in an ounce or two of warm milk, in double the dose, gr. xxx (gm. 2.0).

I refrain from mentioning the drugs of lesser activity classed as sedatives, because their usage is not followed by satisfactory results in such decided spasm as renal colic, and but defers the measures of real value that have just been cited.

Treatment of Other Symptoms Than Pain During the Attack.—Nausea and Vomiting.—This is so intimately connected with the pain, is so secondary in importance to the pain, in the minds of both patient and physician, and so promptly disappears with the pain that our attentions are directed entirely toward the latter. The treatment of the nausea and vomiting itself, except through relief of the colic, would be worse than useless. Those of us who seek for the protective, curative, or compensatory significance of the symptoms in disease will find it in the relaxation of the spasm induced by the vomiting that facilitates the discharge of the offending calculus.

Collapse.—Prostration and collapse may occur in very severe attacks and in patients already weakened. It should be the same as for collapse in other conditions: heat to the extremities, a hot saline irrigation at 110° F., to which a cup of strong black coffee may well be added, diffusible cardiac stimulants, like ammonia, the vasomotor stimulants, adrenalin, m xv-xx, into the muscle, caffein, as a soluble salt, benzoate or salicylate, gr. v (gm. 0.3), or camphor, gr. v (gm. 0.3), in 10 per cent.-20 per cent. solution in sterile oil, or, if the heart is weak, strophanthin, Merck, gr. 1-120 to gr. 1-60. All these may be given hypodermically, or, better, intramuscularly.

Chill and Fever.—This may occur with the colic when there is no infection. It passes with the spasm and needs no especial treatment, unless out of proportion to the pain, when heat to the body during the chill and cool sponge baths during the fever may be given.

Recurring chills and fever occur with infection and will be considered under pyelonephritis.

Suppression.—The simple act of catheterization determines whether we are dealing with retention or suppression. The first 24 hours gives us little alarm, as transitory anurias lasting 12 to 24 hours may occur after an attack of colic, as it may after an operative procedure on the kidney or other abdominal organs, but when it runs beyond the first 24 hours the possibilities of a persistent anuria are greatly increased. There is a curious tolerance, a freedom from any symptoms of distress, that lasts five or six days, that may lure the careless into a sense of security; then come on a feeling of fatigue, a restlessness, sleeplessness, and digestive disturbances that betoken the earliest toxic effects of retained products, then follows a uremia, the patient sinking into a coma in which he dies; rarely are convulsive seizures present.

The mortality of non-operated cases is large, about 70 per cent.

Vigorous treatment in the early stages is indicated, and the earliest signs of poisoning should hasten any surgical procedure of promise.

That many of the kidneys of nephrolithiasis are in a condition of chronic congestion we know, and, appreciating the possibility of an increase of congestion as contributing to the anuria, we apply cups over the kidney region, wet or dry, or hot fomentations. We further relieve the congestion and enhance a vicarious function by inducing vigorous catharsis, by the use of salines and diaphoresis, by the use of the hot pack or hot air bath, or, in the absence of facilities for carrying out these measures, pilocarpin hydrochlorid may be given in doses of gr. 1/10 to gr. 1/5 (gm. 0.006-0.01).

Failure of response of the kidneys to these efforts at relief leads us to stimulate their function by bringing to them normal products of excretion such as water and sodium chlorid, best administered, the water by mouth or with salt as a physiological salt solution by the rectum in the shape of a colon irrigation, or often, better yet, by the drip method of Murphy. One may have recourse even to hypodermoclysis and infusion. With the onset of uremia the infusion may be preceded with benefit by venesection.

Drugs, as diuretics, may be used, my preference being for the theobromin-sodium-salicylate or diuretin in doses of gr. x to gr. xv (gm. 0.6-1.0) three or four times a day, or some other member of the same series, such as theocin, gr. iii-v (gm. 0.2-0.3), 4 i. d., or, in a weak heart, caffein in the same doses. Alkaline salts may prove useful, the citrate, bicarbonate, or acetate of potash or the acid potassium tartrate (cream of tartar) in dose of gr. xv-xxx (gm. 1.0-2.0) every two hours, singly or in combination.

Lemonade or imperial drink (cream of tartar ℥ss to O iii of boiling water, and lemon peel and sugar to the taste) may be given ad libitum. Should the major accidents of uremia, convulsions, occur they are to be treated as in the uremias of nephritis. (See Nephritis.)

Death from suppression usually occurs on the tenth or eleventh day.

After an attack the patient should remain in bed until the ache and soreness have passed and the evidences in the urine of irritation of the stone have ceased.

When infection occurs the picture changes, and we have to deal with pyelitis and pyelonephritis. (See Pyelitis.)

TREATMENT OF THE LITHIASIS

When an attack of renal colic has occurred or the presence of sand, gravel, or calculus has evidenced that a state of lithiasis exists, certain precautions must be observed to avoid an aggravation of the condition or prevent the onset of or recurrence of an attack.

The patient should lead a quiet life with avoidance of excesses of any kind. Violent physical effort or sudden exertion may precipitate an attack of colic. This does not mean that a sedentary life should be encouraged, for this is a factor rather in the production of the condition.

Exercise.—Exercise is important in that a normal functioning of the muscles encourages a normal metabolism in these structures. It must be light in those in whom a colic has occurred, such as walking and calisthenics and massage; may take in more vigorous forms in those in whom sand or gravel alone have appeared, while it may be said that, other things being equal, the younger the subject the more active may be the exercise.

The links in the chain of events that give rise to calculi are not all of them clearly discernible. Our present theories assume that in the intermediary stages of the catabolism of certain organic substances, members of the purin group, or in the play of physicochemical processes in the disposition of certain inorganic salts, such as the phosphates, errors creep in, errors which are determined either by heredity or acquisition; and, further, that damage to the kidney, either a trauma by salts precipitated in its tissue or bacterial infection, plays its part.

It is on this assumption that we order our therapy, endeavoring to improve the functions of the body at large by wholesome living, avoidance of excesses, exercise, fresh air, baths, sufficiency of sleep, and proper correlation of work and rest, aiming to lessen the tendency to errors in metabolism, inherited or acquired. It is on this assumption that we so order our diet as to burden as little as possible the tissues with the substances difficult of catabolism; that we avoid or ameliorate infection, and that we encourage elimination.

Diet.—Our diet is determined by the nature of error in metabolism and that in turn is determined only by the examination of the stone or gravel; hence the great importance of examining each passage of urine to obtain the calculus that may pass.

The vast majority of calculi belong to one of three groups: uric acid, oxalates, or phosphates. Oxalates, looked upon as a product of uric acid with that substance, really form one group, found in acid urines, while the phosphates, found in alkaline urine, form the second.

The phosphatic calculi are as a rule secondary to infection and urinary fermentation, though they may, like the first group, be primary.

Two fundamental principles are to be observed in all cases, (1) simplicity of diet, i. e., avoidance of rich food, multiplicity of courses and foods in a course, and (2) sufficiency and only sufficiency, i. e., for a man of ordinary activities about 40 calories per kilo, or 2,800 to 3,000 calories for the man of average weight.

When the stone or gravel is composed of uric acid, meats should be cut down to small quantities, and especially those containing much cellular material like sweetbread, kidney, liver, or brain; all smoked, spiced, and highly seasoned food and rich pastry should be forbidden.

Fats and sugars should be given in moderation, and the amounts of vegetables, fruit, and milk increased.

When the stone is composed of oxalates those foods that contain much oxalic acid or substances from which oxalates may be derived should be avoided.

Most vegetables (except the legumes, e. g., peas and beans) are interdicted, especially spinach and rhubard, on account of the oxalates they contain and the large amount of calcium in most vegetables, which renders the oxalates less soluble. Figs, strawberries, apples, and pears should be included in this list of articles forbidden.

On the other hand, meats are to be allowed more freely, except the glands, thymus, liver, etc., named above, and, according to Klemperer, gelatin, as all these substances are said to give rise to oxalic acid in the process of metabolism.

When the stone is composed of phosphates the vegetable diet should be decreased and the meat increased.

In many cases the nature of the stone is unknown or it proves to be mixed and, as Klemperer has wisely said, "the prophylaxis of renal calculus lies in the mixed diet dictated by nature," and, indeed, while we seek to be guided by the data of scientific experimentation and research, our deductions should not lead us too far from the road of experience, and we should feel that until our information is very definite it is safer to steer the middle course and not depart too widely from the diet nature has laid down for man.

Alcohol.—Alcohol is to be strictly forbidden. Its ingestion interferes with, not enhances, the metabolic processes.

Water.—That concentration of the urine, i. e., oliguria, facilitates the precipitation of the salts within the kidney substance is a part of the theory of calculus formation, and for this reason as well as for the purpose of flushing out mechanically the small crystals formed before concretion can ensue, water is given and given in abundance. The quantity should amount to 2½ to 3 quarts, which usually affords 48 to 64 ounces of urine.

The water intake should be frequent, every 2 hours, or at mealtime, between meals, and on retiring.

Alkaline Waters.—It is often advised to give alkalies with the idea that they have a solvent effect on the stone formed, which seems to me extremely dubious, but they do act as diuretics and do diminish the acidity of the urine, a reaction which favors uric acid and oxalate formation.

If the urine reacts alkaline the alkali should be intermittent lest the formation of phosphates about existing stone occur. Lithia waters have been much in vogue, but lithia probably has no effect to dissolve the acid calculi present and probably does facilitate the formation of phosphatic calculi. As for the numerous mineral waters advised, one should assure oneself from reliable published analyses that they do contain appreciable quantities of alkalies.

If one wishes the effects of alkalies it is better to use them as such, citrate, acetate, or bicarbonate of potash, in doses of gr. xxx every three hours until the reaction of the urine is reduced to near the neutral point.

As for piperazin and its compounds and other so-called uric acid solvents for which extravagant claims are made, I will express my total disbelief in their efficacy and dismiss them without further comment.

TREATMENT TO DISLODGE THE STONE

Of the various surgical procedures used for the relief of this condition mention only will be made here; for the indications and technique one must consult competent surgical authority.

The X-ray and ureteral catheterization are valued assistants in localizing the obstruction.

Through the ureteral catheter local anesthetics such as cocain or eucain may lessen the spasm and effect a release, or lubricants may be introduced in the same way, such as sterile olive oil or almond oil.

Stripping the ureter through the vagina or rectum and dilatation of the ureter below the point of impaction have been attempted.

Upon surgery we have to depend for relief in a large proportion of the cases. Ureterolithotomy, removal of stone from the ureter, and nephrolithotomy, removal of stone from the kidney, may be done when no infection or complication is present, with a minimum danger.

Of the three operations on the kidney, pyelotomy, nephrolithotomy, and nephrectomy, the first, pyelotomy, or incision into the pelvis of the kidney, is indicated when the stone or stones are of small or medium size and confined to the pelvis of the kidney, and is the operation least to be dreaded and gives the best results.

When the stones are multiple, when branched into the calices and when infected nephrolithotomy may be done. When infection is present, however, the drainage sinus is loath to heal, the infection often persists and thereby endangers the other kidney, so that nephrectomy becomes eventually imperative. When, then, infection is marked, with pyelonephritis, pyonephrosis, and perinephritis, a nephrectomy should be the operation of choice.

A nephrectomy, however, must be preceded by an assurance in the mind of the operator that the remaining kidney is capable of functioning, and entails on him the necessity of determining this fact to the best of his ability by the use of the functional tests, study of water, saline and nitrogenous output, while the intake is accurately known, and by the inspection of the remaining kidney if in doubt.

More serious to the patient and more trying to the therapeutist is the infection superadded to the nephrolithiasis, the treatment of which will be taken up under pyelitis.

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CHAPTER VII

TUBERCULOUS KIDNEY

FRANK SHERMAN MEARA

If, in considering the relationship of kidney affections to tuberculosis, we accept the broad classification of Castaigne and divide the kidney lesions into those which show tuberculous lesions and those which, in a tuberculous subject, may be reasonably attributable to tuberculosis, such as amyloid kidney, parenchymatous or interstitial nephritis, it is only with the first class of cases that this article deals.

Even among the kidneys that show local tuberculosis a discrimination must be made therapeutically between those which share in a general miliary tuberculosis, are bilateral and not amenable to any therapeutic effort, and those which are sometimes called primary, better termed "chronic localized tuberculosis," which, at least in its incipiency, is unilateral in 90 per cent. or more of the cases, and in that very fact enhances the chances of success in treatment immensely.

Of the three possible modes of infection, hematogenous, or descending, by the genitourinary tract, or ascending and by contiguity, it is the consensus of opinion to-day that in the vast majority of cases the route is by the blood stream.

The original site of infection may be obscure, as a gland, a bone lesion, or old pulmonary lesion, or more obvious.

The ureter and bladder may share in the process, while the involvement of the second kidney, so often due to infection through blood or bladder, from the first is an important fact in determining surgical procedure.

TREATMENT

The treatment of tuberculosis of the kidney, like the treatment of tuberculosis of the lung, begins with the earliest possible appreciation of the incipiency of the lesion, so that accuracy of diagnosis becomes an essential part of the treatment, and too much attention cannot be given to the early manifestations of the trouble. The importance of this statement lies in the fact that the only curative treatment follows immediately upon the diagnosis, namely, extirpation of the affected kidney, and the earlier this is effected the more certain may one feel of complete eradication of the disease.

These earliest signs of tuberculosis are usually expressed as disturbances in the urine, either in its composition or its evacuation. So common are these urinary troubles in the experience of every practitioner that a pernicious habit has grown up of classifying such under the rubric of "cystitis" and treating their importance as trivial. So remote are these earlier manifestations from the disasters that eventually make plain the true state of affairs and interrupted by so many periods of remission or amelioration that their connection is often forgotten when the diagnosis is established. When one considers that these so-called cases of cystitis are the announcement of new growths, or stone in the bladder, hypertrophied prostate or of new growth, stone, or tuberculosis of the kidney, and that, even when the condition is true cystitis, pyelitis and pyelonephritis may ensue, one feels that an abnormal condition of the urine or its passage should always be dealt with seriously and investigated in the light of these possibilities.

Perhaps the first symptom that takes the patient to the physician is pain, stiffness or soreness in the muscles of the lumbar region, and here again a besetting sin is the inclination to call all these pains lumbago, forgetting their relation to the kidney and other pelvic organs, as well as to disease of the spinal column and nerve roots.

Again slight constitutional disturbances for which it is difficult to attribute a cause may be due to the involvement of the kidney in a tubercular process.

The disturbances in the evacuation of urine that attract the patient's attention are the quantity, frequency, and discomfort attending it. Polyuria, not necessarily with pain, may be the earliest sign, while frequency of micturition, especially at night, is fairly common. The pain varies in degree and character, but mention is made by some authors of the rather characteristic occurrence of pain in the middle of the act of urination, relieved by its completion.

Blood in the urine may first call the patient's attention to his trouble. Granted that, suspecting the kidney from any of these symptoms, the urine be examined and found abnormal, a systematic search should be instituted for the cause.

The urine in these cases is acid and contains red and white cells. Pus cells in an acid urine excite the suspicion of one of two conditions, a colon bacillus infection or tuberculosis.

Cultures on the ordinary media will discover the bacillus coli, but not the tubercle bacillus, and the absence of growth increases suspicion and leads to a careful search for the tubercle bacillus, which, with care in examination, is almost invariably found. The red blood cells are almost invariably present, intermittently in sufficient quantity to show macroscopically. Such hematuria is always highly suggestive of tuberculosis.

While the finding of the tubercle bacilli may be taken to establish the diagnosis it does not localize the lesion.

A cystoscopy determines the presence or absence of a bladder complication, while the examination of the ureteral orifices may determine the side involved, which a ureteral catheterization makes more sure. The urine taken from these ureters is examined for the tubercle bacilli, blood, pus, etc. The function of either kidney may be determined by this method as well. The X-ray may add its assistance, and in doubtful cases the tuberculin test (hypodermatic) or inoculation of urine into guinea pigs will help.

It is possible that only an exploratory operation will disclose the localization of the lesion.

At the present time the best opinion urges operation at the earliest possible moment when one kidney alone is affected. The failure of satisfactory evidence of cure from medical measures alone, and the danger of increase of the condition with involvement of the other kidney and other structures during the period of medical treatment, urge the surgical procedure. With improvement in surgical technique and diagnostic methods the mortality of the procedure has steadily fallen to 5 per cent. and less in some series, while the percentages of permanent cures have become exceedingly encouraging. Neither the presence of tuberculosis in other organs, nor involvement of the bladder, forbids the operation; on the other hand, the bladder lesion heals the more rapidly after the removal of the kidney.

Involvement of the other kidney does, however, contraindicate nephrectomy. The interpretation of the urine coming from the other ureter in determining the involvement of the second kidney demands at times nice judgment.

Brown (1), quoting Rovsing, says, "the absence of albumin, pus, or bacteria from the second kidney may be relied upon as a rule; if pus and bacteria are present removal of the first kidney is usually contraindicated, while if albumin, but no pus or bacteria, is found, it is not a contraindication, the albumin being due to toxic changes and usually disappearing rapidly after the removal of the diseased organ."

The functional tests carried out are such as were discussed under pyelitis.

As in the surgical treatment of pyelonephritis the question of nephrectomy versus nephrotomy arises, and it seems to me that the pros and cons in the two conditions (see Pyelitis) are much the same, except that hesitation and delay in carrying out vaccination and other medical measures are less justifiable in tuberculosis of the kidney.

If the patient refuse the operation, or if the two kidneys be so involved as to make operation unjustifiable, and, indeed, after an operation, whether there be a residuum of tuberculous infiltration in the bladder or not, the patient is to be treated like a case of pulmonary or other form of tuberculosis. It is generally believed to-day that the so-called primary tuberculosis of the kidney is in reality always secondary to a process elsewhere in the body, as has been mentioned, and even when it is not evident it is to be assumed and the therapy to be determined by the assumption.

Such treatment consists of an abundance of fresh air, a sufficiency of good food, and a proper hygiene. To this may be added cautious vaccine treatment and the relief of symptoms of distress caused by the condition itself or by its complications.

Fresh air has come to be known as the most important factor in the successful treatment of pulmonary tuberculosis, but it is not to be forgotten, as it sometimes is, that fresh air is equally important in the treatment of tuberculosis of other organs than the lung, and, indeed, that in so treating tuberculosis of other organs we are often, perhaps most often, treating an incipient or latent tuberculosis of the lungs. But I think we should go further than that and appreciate that a tuberculosis is not the only infection, acute or chronic, which is benefited by fresh air, as was insisted on in the chapter on Pyelitis.

Patients, then, with renal tuberculosis should have an abundance of air both day and night, and the same devices for sleeping out of doors as are used in cases of pulmonary tuberculosis may be employed here, the porch, the veranda, the lean-to, the tent, the fire-escape, the roof or, failing these, the room with the maximum number of windows wide open and with all the precautions for protection used under such circumstances.

Sunlight, a pleasant environment, quiet, freedom from dust all add their increment to the cure of the patient.

The proper observance of hygienic measures is of importance. Baths should be taken daily not only for purposes of cleanliness, but also as a therapeutic measure.

This latter result is obtained by the cold bath, which stimulates the nervous functions and improves the circulation. In the use of the cold bath, however, one must be guided by the condition of the patient and his reaction toward the procedure. These baths may be taken in the shape of a sponge, plunge, or shower.

The clothing of the patient should be determined by the practitioner, who, appreciating the necessity of avoiding chilling the surface of the body and at the same time preserving the tone of the skin, insists that underclothing shall be of wool, which readily absorbs moisture and, being a poor conductor of heat, allows the skin to cool slowly.

Next to wool silk is preferred for these qualities, while cotton and linen are to be avoided. Overloading with clothes keeps the skin constantly moist in its own secretions and has the relaxing effect of a tepid bath, so that in sudden exposure to the cold air the vessels do not contract and chilling ensues. Clothing indoors should be light and heavier garments added on going out.

A sufficiency of sleep and periods of rest must be afforded; exercise should be insisted upon, but always regulated to the individual patient and his condition and never carried to fatigue. The bowels should be regulated, first by habit and diet and, if necessary, by medication, but always by the mildest that proves effectual.

Diet.—The value of an abundant dietary in pulmonary tuberculosis no longer requires a militant advocacy; it is an accepted fact, but the value of the same dietary may not be equally appreciated in renal tuberculosis, where fear of overburdening the kidney is entertained. When one considers, however, that, of the three foodstuffs, only one makes demands on the kidney, the proteid, and that this may be economized if there is any evidence of incapacity of the kidney in its elimination and, moreover, when one considers the readiness with which compensatory hypertrophy takes place in the kidney, he may dispell such fears and feed abundantly. He should remember that a man whose pursuits are light, such as intellectual work, requires about 3,000 calories of food and about 100 grams of proteid a day and just as much more in the condition under consideration as his appetite dictates and digestion handles and, further, it must be remembered that if our patient is febrile his demands are not less than those of a healthy man at rest, but more—on theoretical grounds his requirements should be as a minimum some 25 per cent. more.

The importance of ingestion of water must not be overlooked, not only as necessary to proper sequence of metabolic events, but also for its specific effect on the kidney and urinary passages.

Tuberculin.—It is tempting to the practitioner to utilize this agent in combating the tubercular process in the kidney, but it must be emphasized that the results obtained so far in this condition do not justify the delay of surgical procedure when the latter is indicated. When, however, operation cannot or may not be performed tuberculin therapy finds a proper place in the treatment of the patient.

It cannot be reiterated with too much insistency that this procedure is not to be lightly undertaken; that it requires thorough knowledge of the rationale, accuracy in technique, and judgment in its application; that one undertaking the use of tuberculin for the first time should give the same conscientious study to the situation he would feel incumbent upon him in approaching his first appendectomy or hysterectomy, for as much harm can come to the patient from an ignorant or neglectful physician in the one case as from an ignorant and neglectful surgeon in the other.

If one cannot do this or has occasion to use tuberculin but rarely it is better to elicit the aid of an expert in its administration, or not use it at all.

SYMPTOMATIC TREATMENT

In nonoperable cases our efforts are directed to rendering the patient as comfortable as possible.

The hygiene and diet are considered as just set down and symptoms treated as they arise. Pain in the kidney region is rarely severe unless complicated with stone or abscess and then is treated with local applications and, if necessary, with drugs in the same manner as specified in the chapters on Nephrolithiasis and Pyelitis.

Still more rarely a hematuria may occur sufficiently severe to demand attention. Rest enforced by morphin hypodermatically is by all odds the most important measure, which may be accompanied by the use of calcium salts, chlorid, gr. xv, by the mouth 4 times a day, or lactate, gr. xx, by hypodermic injection at the same intervals, horse serum in 10-c. c. doses, gelatine foods by mouth to meet theoretical considerations dealing with enhanced coagulation.

Fever is best controlled by the use of cold water, as sponges, and open-air treatment; the chills by local application of heat at the time of their occurrence; sweats, which may be very annoying, by proper care of the body, avoiding too many bedclothes, open air, hot drinks at night, sponges on retiring to which vinegar may be added, ℥i to the pint or more, or alcohol sponges, 25 per cent., or camphoric acid in doses of gr. xx to gr. xxx, or, in most obstinate cases, by atropin in doses of gr. 1-150 to gr. 1-100.

Gastrointestinal disturbances are present, the anorexia taxing the skill of those who order and those who prepare the food, while constipation or diarrhea will require modifications in the diet, the use of laxatives on the one hand, or astringents on the other; but one should always use the mildest of these that are efficacious, cascara, aloin, compound licorice powder on the one hand, and bismuth on the other, before drastic purges or opium are had recourse to.

COMPLICATIONS

In the advanced cases the ingenuity and patience of the physician may be taxed to keep the patient even relatively comfortable in the presence of one or more possible complications. One of the most common is implication of the bladder.

Local applications of heat to relieve pain, the use of acids or alkalies to change the reaction of the urine, belladonna or hyoscyamus to diminish irritability, irrigations of the bladder, or even operation for drainage may be necessary, or the use of opium. Pyelitis and pyelonephritis, more rarely, failure of function of the kidney, with suppression and uremia, demand the efforts specified in the article on Pyelitis or perinephritic or paranephritic abscess requires surgical procedure or may rupture into the peritoneum.

PROPHYLAXIS

Prophylaxis begins with the patient, who submits himself periodically to medical examination or at least on the occasion of slight ailment; then, on the conscientious examination of such patients and the discovering of incipient lesions of a tuberculous nature, the prompt removal of same, if possible, and detailed instruction as to mode of life and work.

Such precautions are doubly imperative in those who are subjected to a tuberculous environment or have a tuberculous history.

REFERENCE

Brown. Osler's Mod. Med.

II. DISEASES OF THE BLADDER

CHAPTER I

CYSTITIS

E. L. Keyes, Jr.

PROPHYLAXIS

The prevention of infection of the bladder is by all odds the most important phase of its treatment, for the normal bladder may be spontaneously infected only by the direct extension of gonorrhea or tuberculosis from a neighboring organ (notably the prostate or the ureter) or by irruption of an adjoining abscess, while the diseased bladder (retention, stone, tumor) may be infected by any pyogenic organisms.

Prevention of Gonorrheal Cystitis.—This is accomplished by the rules, especially the hygienic rules laid down in reference to the treatment of acute urethral gonorrhea. It is to be noted that injections cause acute cystitis only when they are given brutally or to patients who already have a smoldering, unsuspected infection of the bladder neck.

Prevention of Infection by the Tubercle Bacillus.—Tubercular cystitis threatens every patient with tuberculosis of the kidney or of the prostate and seminal vesicles. The proper treatment of the former is nephrectomy, but the latter must be treated by palliative measures.

Prevention of Spontaneous Pyogenic Infections.—The bacillus coli and the pyogenic cocci cannot attack the normal bladder, but if the bladder is congested or otherwise diseased, as is commonly the case in retention, stone, and tumor, it is peculiarly vulnerable to infection. The removal of the etiological factor will, of course, do away with the danger of infection, but such treatment is not always possible, notably in cases of large malignant tumors. Under such circumstances the common sources of spontaneous infection must be carefully watched. These are the prostate and bowel. There is ample evidence to show that constipation causes congestion or inflammatory lesions in the mucous membrane of the colon that permit absorption of the bacillus coli from the bowel. The bacillus passes through the kidney and reaches the bladder, there to cause cystitis if the bladder is not normal. Hence it is of the utmost importance to prevent constipation in such cases.

The second source of spontaneous infection, viz., the prostate, may for years be the seat of a mild infection (usually due to the staphylococcus) without causing any symptoms whatever, yet forming an admirable focus of infection in case of any intercurrent diseases of the bladder itself. The thorough treatment of postgonorrheal prostatitis constitutes the prophylaxis here.

As a further preventive of spontaneous infection it is sometimes useful to employ hexamethylenamin in doses of 20 to 30 grains a day.

Prevention of Catheteral Infection.—We have already insisted upon the gentleness during catheterism, the asepsis before and the antisepsis after, which are required to prevent infection.

TREATMENT OF ACUTE CYSTITIS

Acute gonorrheal posterior urethritis usually extends over into the trigone and is the type of acute cystitis. The treatment of this condition is the treatment of acute gonorrheal cystitis. Acute cystitis from other bacteria usually arises in the course of a chronic inflammation of the bladder, excited by accentuation of the causes which are keeping up the chronic cystitis. Such are the familiar "fit of the stone," the acute cystitis following acute prostatic retention, etc.

The treatment of these conditions, of course, depends largely upon the existing cause. As a general rule, however, it may be stated that rest in bed, catheter irrigations of the bladder once a day with warm silver-nitrate solution (1:10,000), or, if this is too strong, with potassium permanganate (1:6,000), boracic acid, or argyrol. Some alkaline diluent should be administered, and proper measures taken to relieve the cause of the acute attack.

TREATMENT OF CHRONIC CYSTITIS

Since chronic cystitis is simply the manifestation excited by various bladder diseases (e. g., retention, stone, tumor), the first point in the treatment is to decide whether the cause is to be cured or endured. In these surgical days most patients prefer the knife, and this ultimately relieves the inflammation of the bladder. But if the cystitis is to be endured, as in the case of inflammation due to prostatic hypertrophy under treatment by systematic catheterization, inflammation of the bladder must be controlled, whether by the use of one of the bladder washes mentioned above or by hexamethylenamin, or by both. Such treatment will in some cases keep the bladder absolutely clean; but if the bladder is sacculated, or gravely infected, a mild cystitis is likely to persist in spite of treatment.

TREATMENT OF TUBERCULAR CYSTITIS

The treatment of tuberculosis of the bladder, which is never primary, ought to be chiefly directed toward the primary focus, be that in the internal sexual organs or in the kidney. But as in certain cases of genital tuberculosis or in bilateral renal tuberculosis it is impossible to relieve the irritation caused by the primary focus, or if, after removal of the tubercular kidney, ulcerations persist in the bladder, these are best treated by hygiene and injections of tuberculin according to the usual rules.

The curettage or cauterization of tubercular lesions in the bladder is rarely successful, and is likely to leave the patient with a fistula. The treatment is therefore justly looked upon as unwarranted. On the other hand, symptoms may sometimes be relieved by instillation into the bladder every day of 5 or 10 drops of one of the following solutions, beginning at the minimum dose mentioned, and gradually working up to stronger doses: Bichlorid of mercury, 1:20,000 to 1:5,000; gomenol oil, 1 per cent. to 20 per cent.; thallin sulphate, 2 per cent. to 12 per cent.

Rovsing advises that the bladder be washed repeatedly with a 5 per cent. solution of carbolic acid until this no longer returns milky, the treatment to be repeated as soon as the patient has gotten over its effects. He claims excellent results from this treatment, but others have found it excruciatingly painful and relatively inefficient.

As a last resort total cystectomy has been resorted to to relieve the pain of tubercular cystitis.

TREATMENT OF FUNCTIONAL NEUROSES OF THE BLADDER

Three types of neuroses are worthy of consideration:

1. Enuresis.
2. Pain in the bladder or frequent and painful urination due to hysteria or neurasthenia.
3. The same symptoms due to reflex irritation.

Treatment of Enuresis.—Enuresis, or idiopathic incontinence of urine, is that condition in which the patient unconsciously empties his or her bladder without having any control of this act or any physical cause for the same. Enuresis commonly occurs only in children between the ages of three and twelve, and only at night. [Enuresis occurs during the day as well as at night. In the cases most developed it is both nocturnal and diurnal.—Editor.] Inasmuch as this condition is apparently a purely functional one, due to inadequate control on the part of the child, it is usually curable by the removal of any existing cause of irritation, such as adenoids, adherent foreskin, pin worms, etc., plus proper training of the child.

The elements of proper training are:

1. The child shall not drink any fluids after 5 o'clock in the evening.
2. It shall be instructed that the symptom is in some measure under its own control, so that it shall help as far as possible in the cure.
3. It shall be taken up at night often enough to prevent unconscious urination.

At first it may be necessary to awaken the child every half hour and, if so, this should be regularly done for a number of nights, and then the interval gradually lengthened.

Various drugs have been recommended in the treatment of this condition, among which doses of a tenth of a grain of the extract of belladonna, given t. i. d. and increased until the physiological effect is observed, bear the greatest reputation. Rhus toxicodendron, or aromatica, 20 drops of the fluid extract, three or four times a day, is also well spoken of. A few years ago a great deal was thought of Cathelin's treatment, viz.: injection of salt solution into the cauda equina through the terminal foramen of the sacrum, but this is little spoken of latterly.

Among other methods of treatment may be mentioned faradization of the urethral mucosa by means of an electrode in the membranous urethra, with the opposite pole over the pubes; mechanically shutting off the urethra by means of a clip which the patient has to remove in order to urinate, and making the patient lie with head low and pelvis high are worthy of mention. [Faradization, one pole in the rectum or vagina, the other over the pubes, is very valuable, especially in hysterical subjects.—Editor.] But the backbone of the cure is intelligence and hygiene.

Treatment of Reflex Neuroses of the Bladder.—Neurosis of the bladder in the adult is usually due to some functional or organic disturbance of the sexual organs, and the only real difficulty in the treatment of these cases consists in the proper diagnosis of the cause of symptoms.

The causes may be divided into three types:

1. General neuroses.
2. Causes peculiar to the male.
3. Causes peculiar to the female.

Among general causes of bladder pains and irritations, indicanuria, colitis, intestinal adhesions, and phlebitis of the iliac veins are especially worthy of mention.

Among the causes peculiar to the male, prostatitis, vesiculitis, congestion or irritation of the verumontanum, and sexual neurasthenia may be mentioned.

Among causes peculiar to the female are pressure from fibroids or other tumors, exudates and abscesses, uterine misplacement, cystocele, and the pregnant uterus.

We do not include in this list reflex irritation of the bladder from inflammation, or distention of the kidney pelvis.

Having identified the cause of the irritation, its removal, combined with hygiene and a little psychic influence to overcome the bad effect of habit, constitutes the treatment.

Paralysis of the Bladder.—Paralysis of the bladder must be distinguished from atony. Atony is weakness of the bladder wall resulting from urethral retention, of which the common causes are urethral stricture and prostatic hypertrophy, carcinoma, or bar. True paralysis of the bladder may follow diphtheria or may result from any spinal lesion, but the common cause is tabes. Incomplete retention in an otherwise healthy bladder, without urethral obstruction, is often a sign of beginning bladder paralysis, which is the first evidence of tabes.

Treatment in this early stage by salvarsan will sometimes relieve the paralysis, but if retention of urine continues in spite of this, or if the case is seen only after tabes is well established, the paralysis must be treated by emptying the bladder systematically with a catheter as in the treatment of retention from inoperable prostatic hypertrophy, especial care being taken with asepsis and bladder irrigation. If, in spite of such treatment, the patient gets such complications as acute prostatitis, epididymitis, or cystitis much relief may sometimes be afforded by perineal section and drainage of the bladder for a few days through a large tube.

CHAPTER II

NEUROSES OF THE BLADDER

CHARLES GODWIN JENNINGS

PARALYSIS OF THE BLADDER

Paralysis of the bladder is usually a result of organic disease of the central nervous system. Rarely it is a neurosis dependent upon reflected peripheral nervous irritation involving the lower segments of the sacral cord. Any disease or injury of the cord that causes paraplegia may be attended by a vesical paralysis. Dependent upon the nature of the lesion, the paralysis may develop gradually or suddenly, and it may be partial or complete.

Paralysis is to be distinguished from atony of the bladder, a condition more common and met with as a physiological manifestation of the enfeeblement of advancing age or in pathological states, as a result of the muscular debility and degeneration of severe, exhausting acute and chronic diseases. It is true overdistention from atony will cause a temporary paralysis of the muscular wall of the bladder. In this condition, however, the muscular fibers alone, and not the nervous mechanism of micturition, are involved, and it should not be spoken of as a true paralysis.

The striking and important result of vesical paralysis from whatever cause is retention of urine. In all acute and chronic diseases of the nervous system, in comatose states, and in severe exhausting diseases like typhoid fever and other infections the physician should be on the alert to detect a vesical paralysis in its incipiency and before extreme distention has injured the bladder wall and rendered it liable to infection by the colon bacillus or other organism.

Symptoms of retention call for immediate examination of the lower abdomen to detect distention. If the bladder is palpable catheterization is the remedy for immediate relief. The operation should be done under the strictest aseptic precautions. The exaggerated precautions of an abdominal section are none too rigid. So long as the paralysis continues the catheter should be passed every eight or twelve hours, and, in conditions attended by a polyuria, such as diabetes, more frequently. The condition is a serious one, for, sooner or later, the bladder becomes infected and the too familiar train of distressing symptoms follows.

Medical treatment of the condition is of minor importance, although urotropin in full doses will materially aid in preventing or delaying infection. The remedy should be administered as soon as catheterization is necessary. Permanent relief of the paralysis will be dependent upon the course of the primary disease causing the symptom. Remedies directed to the bladder alone are of doubtful utility. Electricity, ergot, and strychnin have been suggested.

ENURESIS

Enuresis occurs in childhood as a *neurosis,* the result of hypersensitiveness of the centers of micturition in the cord, with deficient control by the cerebral cortical centers; or as a *symptom* of various pathological conditions of the urinary and other organs.

The nervous mechanism of micturition consists of: 1. The spinal center in the gray substance of the third, fourth, and fifth sacral segments; sympathetic centers, which are cell complexes inserted in the hypogastric plexus; and cortical and subcortical cerebral centers, which communicate with the spinal centers through fibers in the anterolateral columns of the cord. 2. The efferent limb of the reflex arc, consisting of sensory fibers distributed to the bladder wall, which enter the cord with the posterior roots of the second, third, and fourth sacral segments. 3. The efferent limb of the reflex arc, carrying motor fibers to the sphincter vesicæ and detrusor urinæ, which reach the bladder by the pudendal nerve and the inferior hypogastric plexus.

In infancy urination is purely a reflex act. The urine is retained in the bladder by the tone of the elastic tissues surrounding the neck of the bladder and the urethra, and by the tonic contraction of the sphincter vesicæ. When intravesical pressure reaches a certain point efferent impulses ascend to the micturition center in the cord. From this center efferent inhibitory impulses are sent to the sphincter, causing the muscle to relax, and motor impulses to the muscular wall of the bladder, which then contracts and the bladder empties. At about the end of the second year the spinal reflex mechanism comes under the control of the cerebral centers and within certain limits the urine is retained, or the bladder emptied, at will.

The age at which the cerebral control is established depends upon the inherited nervous stability of the child and to some extent upon the care with which he has been educated. After the third year inability to control the discharge of the reflex spinal mechanism of micturition under ordinary conditions constitutes enuresis. During sleep cerebral control is lessened, and it is at this time that causes that excite reflex discharge are most active. Nocturnal enuresis is the more common form of the disorder. Constant enuresis, diurnal, as well as nocturnal, is frequent. This form may be due to mental enfeeblement or to some serious disease of the central nervous system. Spina bifida occulta is an occasional cause and is incurable. Epilepsy as a cause of nocturnal incontinence should be kept in mind. An incontinence first appearing after the age of five years and showing itself only at irregular times may be of this character. The treatment of cases dependent upon these and other serious diseases of the nervous system belongs to a different chapter.

When it is impossible to ascribe to enuresis an adequate cause, the condition is considered as a neurosis—*essential or idiopathic enuresis.* These cases may be considered as due to the persistence of the infantile mechanism of micturition. With a searching investigation into the clinical history and a complete physical examination, the number of idiopathic cases will diminish, and the physician should not be content with such a diagnosis until all methods of investigation have been exhausted. Still it must not be forgotten that, while in some cases the irritating pathological condition is manifest and would excite an enuresis in a normal child, in most cases the exciting cause is trivial, and the important factor in the condition is the underlying nervous instability of the child.

This nervous instability may be inherited or acquired. The inherited influence is shown by the fact that the parents of these children sometimes give a history of enuresis in childhood and that other children of the family also are afflicted. Other functional nervous diseases, like chorea, habit spasm, and night terrors, may be present.

An acquired nervous instability may be the result of any debilitating acute or chronic disease, or the nervous worry and strain that come with school life. It is quite often noted that an enuresis improves or disappears during the holiday season only to relapse with the beginning of the school period.

Treatment of enuresis should be preceded by a careful, systematic investigation into all the possible conditions that may give rise to it. Treatment directed to this symptom alone, without regard to its cause, usually results in failure. After more or less persistence it is given up, the condition looked upon as incurable by the parents, and the child is left to drift along with a distressing complaint until it is relieved by an accidental cure of the exciting cause or by the complete development of the higher centers of the reflex control that comes with the onset of puberty.

The various diseases that may be the exciting cause of an enuresis, aside from the diseases of the nervous system mentioned above, may be considered under the following heads:

Hygienic.—Bad training, intake of excess of fluid, faulty diet.

Urinary Changes.—Hyperacidity, alkalinity, bacilluria.

Diseases of the Urinary and Pelvic Organs.—Atony of the sphincter, irritability of the bladder, phymosis, adherent prepuce, adherent clitoris, urethritis, cystitis, pyelitis, vulvovaginitis, vesical calculus, intestinal parasites, rectal polyp, anal fissure, chronic interstitial nephritis.

Diseases of Remote Organs.—Adenoids and hypertrophied tonsils, thyroid insufficiency, chronic gastric and intestinal indigestion, chronic constipation, diabetes mellitus and insipidus.

Hygienic.—A child must be taught to control its bladder and to make known its desire to empty it. Carelessness in this regard may result in a prolongation of the habits of infancy. Properly trained, the child should have the mechanism of micturition under cerebral control by the end of the second year.

A faulty diet will be found to be the determining factor in many cases in which the enuresis first appears after the age of four or five years. Dietetic errors produce incontinence in predisposed children by inciting chronic intestinal indigestion with resultant absorption of the products of excessive intestinal putrefaction and fermentation. Constipation, flatulence, an irritating urine, together with the exaggerated irritability of the reflex center, resulting from impaired nutrition, determine the incontinence. A diet in which carbohydrates, particularly sugar and sweets, are in excess is the most frequently observed fault. The writer has seen many cases promptly cured by a reduction in the starch and sugar content of the diet. Elimination of the sugar alone is sometimes successful. Excess of meat proteids is more rarely a cause. In every case of enuresis the diet should be carefully prescribed. There should be such an adjustment of proteids, fats, and carbohydrates as to bring about the best nutrition of the child with the least tax upon the digestion. Excess of fluids should be avoided, although it is useless to attempt to control an enuresis by restricting the fluids to a quantity below the physiological requirements. The food should be bland, and all stimulating articles, like tea, coffee, spiced and seasoned foods, and meat juices, should be forbidden.

Urinary Changes.—Vesical irritability may be caused by polyuria, hyperacidity, or alkalinity of the urine, and bacteriuria. A careful analysis of the urine, chemical and microscopical, should be a routine procedure in the examination of every child with enuresis. The necessity for this cannot be emphasized too strongly. The routine tests for albumin and sugar are not enough. A hyperacidity, a few cells, or an excess of epithelium, indicating a mild vesical catarrh, or infection with the colon bacillus may be the exciting cause of an enuresis and escape detection unless carefully sought for.

Polyuria may result from too much fluid in the diet, as noted above. Diabetes mellitus will be revealed by the urinary analysis. Chronic interstitial nephritis, although a rare cause of nocturnal polyuria with incontinence, should always be kept in mind and carefully excluded.

Hyperacidity of the urine, with heavy deposit of urates, uric acid, or oxalate of calcium, is associated with many cases of enuresis and may be an important factor in its production. This condition may attend chronic derangements of digestion, or an excess of meat proteid in the diet. Such cases demand a careful diet prescription with an alkali: acetate or citrate of potassium with tincture of hyoscyamus.

Alkalinity of the urine occasionally is a cause of vesical irritation. It occurs with a polyuria and a urine of low specific gravity, which deposits triple phosphates and may contain a trace of albumin. Guthrie states that a characteristic of this condition is the quick repetition of the discharge of a large quantity of urine after the child has been awakened to empty the bladder. This condition may be the urinary manifestation of an excess of starch or sugar in the diet.

Microscopic examination of the stained centrifuged sediment of the urine shows that infection of the urinary tract by the colon bacillus is an occasional cause of enuresis. The urine, when passed, is clear, highly acid, has an offensive odor, and stains the clothing. On standing, it quickly becomes turbid, and its reaction changes. There may be a trace of albumin. This condition may escape detection because of imperfect examination, and the failure to appreciate that infection may be present without pus in the urine.

The treatment of colon bacilluria is the administration of full doses of alkali with or without urotropin. Five to ten grains each of acetate and citrate of potassium, well diluted with water, may be given before each meal and at bedtime. Larger doses may be given in obstinate cases. At the same time five grains of urotropin may be given after each meal. Tincture of hyoscyamus or belladonna in elixir of buchu may be given at night to lessen the irritability of the bladder. This treatment should be kept up for several weeks or the alkali and urotropin may be alternated. At the same time the bowels should be kept free and the colon flushed two or three times a week to prevent reinfection. While rarely necessary in a simple bacilluria, in obstinate cases the administration of a colon vaccine after the method spoken of under cystitis and pyelitis may be advisable.

Diseases of the Urinary and Pelvic Organs.—Atony of the sphincter is a condition not easy to demonstrate, but may exist in feeble children, convalescent from acute illnesses, or suffering from chronic nutritional diseases. In this condition the urine is frequently voided in small quantity both day and night. It probably never exists alone. These cases may be relieved by the general measures recommended below, directed to improving the tone of the muscular and nervous systems, and by local treatment to the sphincter itself. The faradic or galvanic current finds its use here. The application may be made with one pole attached to a large flat electrode placed over the lumbar or suprapubic region and the other attached to a small electrode applied to the perineum. Some cases do better with a vaginal, urethral, or rectal electrode. Care must be used to begin with the galvanic or a very mild faradic current, and gradually accustom the child to it. The discomfort and fright produced by an injudicious application at first may make further treatment impossible. In some cases massage of the neck of the bladder through the rectum may have a marked tonic effect on the sphincter, and at the same time relieve a catarrh of the deep urethra and prostate.

Excessive irritability of the bladder may be of nervous origin, but usually it is a symptom of infection along the urinary tract. The irritability may persist after an infection of the urinary tract or some remote cause of reflex irritation has disappeared. Many cases of enuresis in which it is impossible to find an adequate cause may be explained in this way. The cause has been removed but the irritability that resulted from it remains and the bladder is excited to contract by the pressure in it of a small quantity of normal urine.

The treatment of an irritable bladder after the exciting cause of an enuresis has been removed consists in the administration of vesical sedatives and the education of the bladder to endure greater distention. It is often of advantage to keep the child in bed for a few days and instruct it to void at gradually increasing intervals. In cases that have persisted for some time and resist other measures the injection into the bladder of increasing amounts of a sterile salt or boric acid solution will be followed by good results. The injection may be made through a small soft catheter, or, in older boys, by hydrostatic pressure without the introduction of the catheter.

Urethritis, cystitis, and pyelitis are revealed in the urinary examination and by pain and other symptoms referable to different portions of the urinary tract.

Urethritis is an uncommon cause, although it may be present in older boys with a long-continued enuresis, or as a result of bladder infection by the colon bacillus. Very rarely there is a gonococcus infection. A deep urethritis in older boys may require the passage of a steel sound or the injection of argyrol or silver nitrate into the deep urethra. The well-known technique of modern genitourinary surgery should be used. These measures sometimes promptly cure an enuresis.

In addition to the administration of urotropin and the salts of potassium, colon infection of the bladder and pelvis of the kidney may be treated by a colon vaccine. A stock vaccine, or, when possible, an autogenous vaccine, may be given. Ten millions is a suitable dose for a child of five years. This may be repeated at intervals of four days for several doses, and the number increased to fifteen millions. The results of vaccine treatment of colon infection of the urinary tract are often brilliant.

A vesical calculus should be in mind in certain obstinate cases. An elongated and adherent prepuce and phimosis in boys, or an adherent clitoris in girls, is often present in enuresis, and is the local irritant that maintains the hyperexcitability of the micturition reflex. They should always be sought for, and, if present, relieved. Circumcision should be performed in cases with redundant prepuce and phimosis. Harmonic has reported 187 cases of enuresis treated by circumcision, of which 130 were cured, and many other writers speak with equal confidence of its beneficial effect.

According to the writer's experience, the results of operation alone will often be disappointing, and too much should not be promised from it. Phimosis should be looked upon as one of the factors, sometimes the most important, of incontinence. Even when circumcision is performed, treatment for the neurosis will usually be necessary to complete the cure. The pathological irritability of the nervous mechanism of urination that has been set up by the prolonged local irritation will require careful treatment on the general lines laid down elsewhere. Without circumcision, however, all efforts to overcome the condition may be unavailing.

A persistent balanitis or a vulvovaginitis may be the exciting cause of an enuresis, and certain cases may be traced to irritation of the lower intestinal tract. Parasites, particularly the oxyuris vermicularis, should be sought for and properly treated. A rectal polyp or an anal fissure is a rare exciting cause. Constipation is a common complication, and may be an important etiological factor. The influence of a loaded rectum may be readily noted in mild cases. Here it will often be noted that the incontinence relapses or is aggravated by a period of sluggish bowels. Relief of constipation should be a part of the routine treatment of every case. When the relation between the two symptoms is marked the thorough emptying of the rectum before bedtime may alone cure enuresis. In the absence of mental deficiency, cases showing diurnal as well as a nocturnal incontinence are usually due to some severe irritation along the urinary tract or the contiguous structures. Dysuria also points strongly to a local cause.

Diseases of Remote Organs.—Children with adenoids and hypertrophied tonsils are more liable than others to suffer from incontinence. Fisher found enuresis in 106, or 14.8 per cent., of 716 cases operated upon, and several other writers have reported about the same average percentage. Occasionally the enuresis is due solely to this cause, and operation will promptly be followed by relief. More often this is only one of the factors or a complication having no etiological significance. As in circumcision, the physician should be careful not to promise a cure by operation alone. Too frequently removal of the adenoids has no influence on the cause of the enuresis. Such a result is discouraging to parents who have been assured of benefit and may lead to the abandonment of other essential therapeutic measures.

When a cause of well-marked respiratory obstruction, adenoids should be removed. The partial asphyxia produced by them during sleep stimulates the vasomotor center in the medulla, and excites a polyuria, an important factor in many cases of incontinence.

One of the most interesting of recent observations is the curative effect of desiccated thyroid in certain cases of enuresis. Hertoghe, of Antwerp, first saw beneficial results from its use. Leonard Williams, of London, reported 25 cases, only one of which failed to be benefited. The treatment was suggested to him by the fact that a boy operated for tonsils and adenoids for the cure of enuresis was made worse. Reasoning that the removal of the adenoids and tonsils had deprived the boy of a necessary internal secretion, thyroid was administered to make up for the loss. The boy was promptly cured.

Ruhräh treated a small series of cases and noted remarkable results in children presenting signs that might be attributed to thyroid insufficiency.

The writer has not had an opportunity to try out this remedy, except in two cases, both girls from four to five years old, with intractable incontinence from infancy, but without symptoms to indicate thyroid insufficiency. They were not influenced by careful thyroid medication extending over a period of three weeks.

Various disturbances of function of different organs in the adult resulting from thyroid insufficiency are so often markedly benefited by thyroid feeding that the experience of Williams should be given serious attention.

As described by Williams, children with thyroid insufficiency are undersized and under weight. Their vitality is low and they continually show a temperature one or two degrees below the normal. They complain of cold, even in summer, and often have cold, blanched fingers. Many of them have adenoids and show a high-arched palate. Mental sluggishness is not characteristic. Such cases and others of obscure etiology in which well-directed therapeutic efforts have failed may be experimentally treated with thyroid. To a child of six years one-half grain of desiccated thyroid may be given morning and night, and the dose increased gradually to one grain three times a day. It is better to begin with a small dose, as overdosage has caused in some cases an aggravation of the symptoms. Favorable results should come within two or three weeks, and it is unnecessary to continue the remedy beyond this time if no effect is produced. According to Williams, cases favorably influenced by the thyroid often show quite a remarkable improvement in nutrition and a large gain in weight.

A chronic gastric or intestinal indigestion is a predisposing rather than an exciting cause of an enuresis, although it may act in both ways. The impaired nutrition, deranged metabolism, and anemia that result from indigestion render the reflex centers highly sensitive to irritation and make possible an enuresis from an insignificant local irritation; or the pathological changes in the alimentary tract may in themselves act as the reflex irritant that excites the involuntary emptying of the bladder.

General Treatment

All cases of enuresis will require systematic therapeutics of a general character in addition to the measures instituted directly for the relief of the specific etiological factor, if one can be found. The condition is so complex, so often the result of a combination of causes, that it is best to take advantage of all means to diminish the hypersensitiveness of the mechanism of micturition.

Certain routine instructions should be given for all cases. While punishment in cases that have continued for any length of time is universally admitted to be more harmful than otherwise, certain careless and lazy children may well receive judicious discipline. Moral treatment, an appeal to its sense of delicacy, and some system of rewards will materially assist the child's cerebral control. A cold spinal douche with a brisk rub may be given on retiring and the rectum unloaded by a warm oil enema. The child should be awakened late in the evening to empty the bladder. The room in which the child sleeps should not be too cold, and he should be plentifully supplied with bed clothing. Elevation of the foot of the bed may be of use, but is often impracticable. Some mechanical device to prevent the child from lying on the back has occasionally been successful. An electrical device composed of two metal disks separated by absorbent cotton or some fabric connected with a battery and bell has been found successful by Genouville. Saturation of the fabric connecting the disks with urine completes the circuit and the bell rings. Devices of this kind probably act by suggestion or by putting the cerebral centers on the alert for the impulses from a full bladder.

Every effort should be made to give these children quiet, invigorating surroundings. A life in the country is to be selected whenever possible. The nervous tension of school life should be relieved or ameliorated, and all exciting occupations and amusements forbidden. A holiday at the seashore, with freedom from the restraints of conventional city life, is often sufficient to effect a cure. Restriction of fluids in the latter part of the day has a good effect. No fluid except what is contained in the food should be allowed after four o'clock in the afternoon, and broths, milk, or other liquid foods should be limited to the lowest possible quantity.

Careful inquiry should be made into the diet habits of the child, and every article that may be a source of irritation to the urinary tract excluded. As above noted, sugar products and highly seasoned food are often the unsuspected offenders. A simple, plain, but well-balanced and digestible diet should be prescribed. Gastric and intestinal indigestion should receive the most approved treatment.

Many children with enuresis are robust, well nourished, and otherwise well. Here it is unnecessary to give tonics or otherwise to burden the parents with useless restrictions. Local irritation of some nature is the cause of the trouble and should be diligently sought and remedied.

An undernourished and anemic child should be built up by iron, arsenic, and cod-liver oil, with rigid attention to its food and digestion. Enuresis and night terrors often go together, and reveal the nervous origin of the trouble. Bromid of sodium, five to ten grains, before supper and at bedtime may act beneficially. Chorea should make the physician suspect a rheumatic cause and would suggest salicylate of sodium.

Of drugs that directly influence enuresis, belladonna or atropin is the most frequently successful. The drug apparently owes its beneficial influence to its sedative effect on the muscular wall of the bladder. It may be given in the form of tincture of belladonna or atropin sulphate. Tincture of belladonna may be given to a child of five years of age in a dose of five minims three times a day, and increasing the dose by one minim every second day until flushing of the face from twenty to thirty minutes after administration shows that the full physiological effect has been obtained. It may then be reduced one minim a day until the flushing ceases, and this dose continued for several days. If no effect is produced after the drug is pushed to the limit, it is useless to continue it. It is unwise to keep a child for weeks in a state of mild belladonna intoxication. Atropin sulphate may advantageously take the place of belladonna. A common way to prescribe it is one grain to two ounces of water. One minim of this solution will represent about 1-1,000 gr. of the drug. Two minims may be given as the initial dose to a child of five years, increasing one minim every second day, as directed for the administration of tincture of belladonna. The writer prefers to give the remedy in a vehicle such as elixir of buchu or santal, with five to ten grains of acetate of potassium, which acts to allay irritability of the mucous membrane of the bladder. Tincture of hyoscyamus may replace the belladonna when there is infection along the urinary tract. It may be given in a dose double that of belladonna. Tincture of nux vomica or strychnin sulphate is indicated in all cases in which an atony of the sphincter is a factor in the enuresis. Strychnin acts also as a tonic to the hypersensitive centers in the cord. Freyberger, in cases that have resisted belladonna, has found good results from fluid extract of rhus aromatica in doses from five to ten minims to a child of five years. He believes the drug acts as a powerful tonic to the bladder, similarly to nux vomica. In cases of nocturnal incontinence the doses of belladonna may be given in the latter part of the day so that the child will be under its full influence during the sleeping hours and thus escape some of its unpleasant effects.

Ergot has been given as a vesical tonic in cases of atony of the sphincter. In the writer's experience it has not proven of value when belladonna and strychnin have failed.

Epidural injections of saline solution were proposed by Cathelin in 1901 to relieve incontinence. The method may be used in some obstinate cases when gentler measures have failed. The writer has not seen cases where he has considered it justifiable.

Under aseptic precautions a hypodermic needle two and one-half inches long is introduced through the sacral hiatus at the lower end of the spinal cord. It thus reaches the space between the periosteum lining the canal and the dura mater of the cord, below the filum terminale which ends at the second sacral vertebra. The formula recommended is sodium chlorid, 0.2 gram; cocain hydrochlorate, 0.01 gram; sterile distilled water, 100.0 grams. Ten c. c. of this solution should be slowly injected, gradually increasing the dose in succeeding injections to 20 c. c. Five to ten injections are given at intervals of from two to five days. A mild anesthesia may be necessary in nervous children.

Tuckey, Bernheim, Cullor, and other practitioners of hypnotic suggestion consider this form of treatment almost specific in enuresis uncomplicated by organic disease, and have reported some remarkable collections of cases. Dubois would reserve hypnosis for exceptional cases, although he believes that suggestion is the most important part of a number of methods of treatment. Considering the confidence with which some writers speak of the different means of cure which they advocate, it seems quite probable that the results obtained are often largely due to the influence of suggestion upon the child. A physician confident of the results of his favorite measure unconsciously brings to bear a strong suggestive influence that is an efficient aid in restoring the nervous balance of a pathological reflex mechanism.

Hypnotic suggestion may have a use in selected cases that resist well-ordered conventional therapeutics, but its field is necessarily restricted. Very few physicians possess the mental and physical characteristics that are to be noted in successful practitioners of suggestive therapeutics. Toward hypnotism most of them feel, with Dubois, that "the attitude of the wonder-worker that one has to take is so repugnant to me that it brings the blush of shame to my cheeks when I decide to use it."

III. DISEASES OF THE MALE SEXUAL ORGANS

CHAPTER I

GONORRHEA

E. L. KEYES, JR.

The underlying principles for the treatment of acute urethral gonorrhea are quite different from those upon which the treatment of chronic urethral gonorrhea is based. And since our practice upon so rebellious a malady depends very largely upon the soundness of our principles it is essential at the outset that we define what is meant by acute and what by chronic urethritis.

Acute urethritis is an inflammation of the urethra of such severity as to cause pain, bleeding, or a profuse discharge of pus (exception made for the obviously chronic conditions about the posterior urethra, and its adnexa, that so commonly cause painful erections and painful urination, though manifestly not associated with any acute inflammation).

Chronic urethritis, on the other hand, is that inflammation of the urethra characterized by slight or intermittent discharge of pus, no pain (with the exception just referred to), and no spontaneous hemorrhage, unless this hemorrhage be due to stricture.

Acute gonorrheal urethritis usually lasts at least a month from the onset of the disease, for, although the acuteness of the inflammation may be controlled during this first month by the use of the so-called repressive treatment, the patient is, nevertheless, in a state of what might be called potentially acute gonorrhea, since, during this period, the gonococci reside in a mucous membrane not fortified by local immunity, and are, therefore, capable of exciting an acute inflammation if ever the repressive treatment fails, either through excessive zeal or through neglect.

Later in the disease, at any time while there are still gonococci in the urethra, an acute outbreak may occur, but this exacerbation differs from the original outbreak by its lesser severity, both in intensity and in duration. Moreover, subsequent attacks of gonorrhea are likely (doubtless because of local immunity) to be milder, briefer, and more readily controlled than the first outbreak. This is a fact always to be remembered in treatment.

Chronic urethral gonorrhea is divisible into three periods:

1. That of true chronic gonorrhea, during which the gonococci are still present.
2. That of postgonorrheal urethritis, during which a catarrh persists, although the original cause of that catarrh, i. e., the gonococcus, has disappeared.
3. The period of postgonorrheal neuroses.

Needless to state, the duration of these conditions knows no precise limits. The postgonorrheal urethritis is usually brief; while the postgonorrheal neuroses differ in no respect from similar neuroses due to purely sexual causes.

TREATMENT OF ACUTE URETHRAL GONORRHEA

The treatment of acute urethral gonorrhea comprises five distinct topics:

The preventive treatment, the abortive treatment, the repressive treatment, the terminal or expectant treatment, the treatment of complications.

Preventive Treatment

Gonorrhea in the male may be prevented by discouraging the male from visiting the foci of disease, by disinfecting those foci, or by protecting the errant male by the use of a condom, or of preventive injections after exposure.

The prevention by social and moral means, by educating the young in a knowledge of their sexual instincts, and the dangers arising therefrom—this is at present receiving a world-wide trial. The elucidation of sexual matters in an intelligent and sympathetic way to adolescents can scarcely fail to prove a most efficient deterrent in many instances. And although generations will doubtless pass before the result of the crusade can be known this is surely the safest way of preventing gonorrhea.

Prevention of gonorrhea by disinfection of the foci of disease is practicable only in reference to the army and navy services, where the men, as well as the women, can be kept under observation. Reglementation in the world at large, where most women and all men are clandestine offenders, and, therefore, escape the law, has proved quite inefficient.

Personal prevention by the use of a condom is not as safe as it would appear to be, for I have known several men to become infected in spite of an unbroken condom. Personal prevention by the use of antiseptic injection immediately after exposure is singularly efficacious.

Statistics of the navy show a reduction in venereal morbidity by the injection of 1 per cent protargol (or a similar antiseptic) applied within 12 hours of exposure and retained for three minutes.

Irrigation with 1:5,000 potassium permanganate is apparently equally efficient. The private citizen may protect himself by the injection with a minim dropper in the first inch or two of the urethra of a few drops of 5 per cent. protargol in glycerin, by the instillation of a protargol bougie, or by the injection of a 1 per cent. solution of the same. Of these methods the most efficient is probably the last, if the solution is freshly made from a powder (which can be conveniently carried about). Many other silver salts, if used in appropriate strength, are doubtless as efficient as protargol. The earlier the injection is made the more efficacious it is. The injection should never be repeated.

The Abortive Treatment

The attempt to abort acute gonorrhea depends for its success upon the application to the urethra of a solution sufficiently strong to kill all gonococci in one, or, at most, in a few, applications. Although such methods may abort many cases, they are calculated to excite an acute urethritis, so that, if the last gonococci are not killed, their multiplication is encouraged, and failure implies a much more severe acute gonorrhea than if the patient had been treated from the outset by repressive measures.

In practice one aborts fully as many gonorrheas by repressive measures as by the abortive treatment, and one leaves those not aborted in a far better condition. For this reason I do not employ, and cannot advise, the use of abortive treatment.

Repressive Treatment

The repressive treatment of acute urethral gonorrhea consists in the employment of all such measures as are calculated to destroy the gonococcus without irritating the urethra. The keynote to this form of treatment is the avoidance of all irritants. Repressive treatment is both general and local.

General Treatment

Cleanliness.—Inasmuch as any contact of the urethral pus with the patient's eyes is likely to excite virulent gonorrheal ophthalmia, he must be impressed with the necessity of cleansing his hands very carefully every time he touches the penis. For this cleansing no antiseptic is required; soap and water suffice. It is noteworthy that infants acquire gonorrheal ophthalmia with the greatest readiness; adults rarely do so. In almost 15 years of office experience I cannot recall a single case of conjunctival infection among the patients who came to me with urethral gonorrhea.

The local cleanliness required applies chiefly to those patients whose discharge is free. If the foreskin is long, especial care must be taken in cleansing the preputial cavity, and hydrogen peroxid (diluted to one-third strength) may be required for this.

In order to keep the urethral discharge from reaching the clothes and irritating the foreskin, a protective dressing must be worn until the discharge has been reduced to a morning drop. The best dressing for a patient with a long foreskin is a strip of a 2-inch gauze bandage perforated to admit the glans penis. The gauze is slipped on back of the corona and then the foreskin is carefully pulled forward, holding the gauze in place. If the discharge is not profuse, a piece of cotton may be employed instead of this gauze. While, if the foreskin is short, the patient has to protect himself by wearing one of the so-called gonorrhea bags, or some home-made substitute for the same.

Rest.—Physical rest is one of the most important helps in the successful treatment of acute gonorrhea. It is impracticable to put the patient to bed. Indeed, it is questionable whether the mental disturbance excited by complete rest, with all the social and physical inconvenience this entails, would not do more harm than good. But, while the patient is permitted to be up and about, his business or pleasure should not include prolonged standing; any but the shortest walking; or riding in railroad trains or automobiles. All forms of exercise are prohibited.

The young man often finds this physical inactivity one of the most trying features of treatment, and it should be the physician's aspiration to relax his prohibitive rules as soon as this is possible.

A suspensory bandage, or jock-strap, should be worn throughout the acute stage of gonorrhea, for the prevention of epididymitis.

Diet.—The rigorous diet usually prescribed excludes all alcohol, spices, rich and indigestible sauces and foods, fruit, coffee, tea, and sparkling water.

I have found it of no benefit to the patient's urethra to be so strict, and a great encouragement of his mind to permit a greater latitude. Alcohol, spices, and condiments must, of course, be prohibited, and it is well to specify ale, beer, cider, and ginger ale, besides insisting that any substance which burns the palate as it enters the body will burn the urethra as it issues forth (we speak, of course, of chemical, not of physical, heat). Indigestion, whether from overeating or from indiscreet eating, is harmful, and fruits, especially lemons and grape fruit, as well as asparagus, are apparently irritating, unless eaten in moderation. But there is no reason to prohibit these absolutely nor to prohibit tea or coffee at all.

Sexual Hygiene.—Absolute continence is essential, in thought as well as in act, throughout the acute stages of the disease.

Diluents.—Most patients should drink as much water as they can. But the exceptional man, whose capacity for water is unlimited, must be restricted to that amount which causes him to urinate every two hours; by exceeding this he would only irritate the urethra. If the patient can afford it, it is preferable that he drink an alkaline water, and, of these, Vichy Celestins is the best.

Internal Medication.—The drugs usually employed in the treatment of acute gonorrhea may be classified under 4 heads:

1. Urinary antiseptics.
2. Alkalies.
3. Demulcents.
4. Anodynes.

URINARY ANTISEPTICS.—Urinary antiseptics, such as hexamethylenamin, methylene blue, salol, and the benzoates, have no beneficial effect upon acute urethral gonorrhea, and should not be used in this condition unless for the treatment of acute pyelonephritis complicating the gonorrhea. The use of salol (in the form of the compound salol capsule) is harmless, but does no good; but hexamethylenamin, though frequently employed, is certainly useless, and may be harmful to those whose urethræ are sensitive to this drug.

ALKALIES.—Alkalies are employed in the treatment of acute urethral gonorrhea, on one of two theories: either that (1) the urine is overacid and irritating, or that (2) the gonococci thrive more upon an acid than an alkaline medium, and therefore the administration of alkalies directly attacks the gonococcus.

In times past I have inclined to the former theory, and have, therefore, administered alkalies only for the purpose of neutralizing an overacid urine. But further observation has convinced me that this is not enough; it is better that the urine should be alkaline rather than neutral; and therefore alkalies should be administered to all cases. In private practice the desired result may usually be obtained if, in connection with repressive local treatment, the patient drinks a quart or two of Vichy Celestins, or takes 15 or 20 grains of sodium bicarbonate before each meal. But, in the clinic where a repressive treatment is relatively unsuccessful, or, in any case, if the local repressive treatment fails and the urethral inflammation continues intense, it is better to give one of the stronger alkalies.

Potassium Citrate, Potassium Acetate, Liquor Potassæ.—These are the favorite urinary alkalinzers. The dose of each is 5 to 15 grains (0.3 to 1.0 gram). They are usually given in combination with one of the prescriptions detailed below. The acetate is the most diuretic, but also the most difficult to digest. I therefore prefer the liquor potassæ or the citrate.

DEMULCENTS AND BALSAMICS.—The demulcents, such as buchu, pareira brava, uva ursi, etc., were much depended upon by the passing generation of physicians in the form of decoctions, the benefit derived being in proportion to the amount of water drunk with them; but they are no longer generally employed.

The balsamics, however, have a definite though limited value. Many patients do fully as well without balsamics as with them, but for others the effect of these drugs is manifest. They seem especially effective, however, in the treatment of nongonorrheal urethritis, which is often promptly cured by balsamics without any other treatment. It is, therefore, probable that the chief effect of the balsamics is upon the mucous membrane rather than upon the gonococcus, and inasmuch as so many cases of gonorrhea do perfectly well without them it is obviously wiser not to administer them in doses large enough to upset the digestion or to cause any symptoms of poisoning. Since even small doses disagree with some stomachs, it seems preferable, if the patient cannot take with comfort a balsamic in the minimum dose here set down, that no effort should be made to administer it at all.

It seems unwise to attempt a discussion of the virtues of the many proprietary preparations of balsamics which are constantly appearing upon the market, supported by enthusiastic theorists and scientific claims. I have experimented with many of them, and have never found that they possessed any peculiar virtues. Therefore, while we may still hope for the appearance of a synthetic balsamic, singular both in its efficiency and digestibility, these need not delay us at present.

Among the older balsamics, four merit especial mention. These are sandalwood oil, copaiba, wintergreen oil, and cubeb.

Sandalwood Oil.—The oil of yellow sandalwood is probably the best of balsamics for most cases. It is administered in soft capsules, containing 5 or 10 minims (0.3 to 0.6 c. c.). The dose should be at least 5 minims (0.3 c. c.) three times a day. It is better to give 10 minims (0.6 c. c.), and few patients can take more than this with impunity. A favorite prescription combining alkalies and balsamics is the following:

℞ Potass. citrat., 8.00-25.00 gm. (ʒij-vj).
Ol. santal, 15.00-25.00 gm. (ʒiv-vj).
Syr. acaciæ, 30.00 (℥j).
Aquæ menth. pip. q. s. ad 100.00 (℥iij).
M. Shake.
Sig. Teaspoonful in water two hours after eating.

Copaiba.—The balsam of copaiba is probably equal in efficiency to the oil of sandalwood, but it is much more difficult to digest. The dose is the same.

It is usually prescribed in capsules either alone or in combination with other balsamics, as in the so-called salol compound.

℞ Phenylis salicylatis, gr. iii (0.2 gm.).
Copaibæ, m. x (0.6 gm.).
Cubebæ, m. v (0.3 gm.).
Pepsinæ, gr. i (0.065 gm.).

Copaibal poisoning shows itself both by disturbance of the digestion (nausea, vomiting, etc.) and by toxic erythema, which usually consists in closely aggregated, slightly red blotches plentifully scattered over the trunk. The eruption itches and is readily cured by discontinuance of the drug and the administration of a laxative, and, if necessary, of warm baths containing 3 oz. (100.00 c. c.) of sodium bicarbonate to 10 gallons of water. This eruption, if severe, may be accompanied by fever and temporary subsidence of the urethral discharge.

Wintergreen Oil.—Wintergreen oil, whether natural or synthetic, is given in 10-minim (0.6 c. c.) capsules. It is not as efficient as either sandalwood or copaiba, but may be given in a dose of 10 minims (0.6 c. c.) three times a day to those patients with whom these balsamics disagree.

Cubeb.—This drug is also relatively inefficient, but may be given as a substitute for the stronger balsamics. I fancy it is rarely employed nowadays, except in the salol capsules already mentioned.

ANODYNES.—*For Chordee.*—For the painful erections and chordee of acute gonorrhea the best treatment is preventive. Erections may be minimized by sleeping with few covers and eating lightly at the evening meal. If they occur they may be relieved by urinating or by immersing the penis in cold water.

Drugs are singularly inefficient, although the following are recommended to be taken on retiring: Sodium bicarbonate, 15 grains (1 gram); lupulin, 30 grains (2 grams); codein, or the somnifacients, such as trional or veronal.

For Painful Urination.—If the pain is due to injections, these must be discontinued; if to inflammation of the posterior urethra, the presence of prostatic abscess must be suspected.

The following anodyne mixture is often useful:

℞ Liquoris potassæ, ʒij-vj (8.00-25.00).
Tincturæ hyoscyami, ℥ss-j (15.00-35.00).
Aquæ cinnamomi q. s. ad ℥iij (100.00).
M.
Sig. Teaspoonful in water two hours after each meal.

An appropriate dose of codein phosphate or the bromids may be added to this prescription.

If the pain is terminal and due to spasm at the close of the act of urination, this may be minimized by instructing the patient not to empty his bladder completely, but let the last of the urine dribble away without the distressful piston stroke. The instruction is hard to follow but may afford great relief.

Hot water is valuable in various ways. The pain of urination may be modified by soaking the penis for a few moments in water as hot as can be borne and urinating into this.

Hot sitz baths are sometimes more efficacious, and, above all, hot rectal douches, as in the treatment of posterior urethritis.

The other measures mentioned under the head of local treatment of posterior urethritis are, of course, to be employed.

Instructions to Patients

Of late years the commendable practice has arisen of distributing to dispensary patients suffering from venereal diseases a card indicating the chief dangers of the disease and the precautions they personally must take to encourage speedy cure and to protect their fellows. The following list for this purpose is copied. with but few minor changes, from that employed by Follen Cabot:

INSTRUCTIONS TO THOSE HAVING GONORRHEA, OR "CLAP"

Gonorrhea, or "Clap," is a contagious disease which requires treatment until the physician pronounces you cured.

To avoid infecting others, and to prevent complications, such as stricture, swollen testicles, etc., the following rules should be observed:

1. During the first few weeks walking should be limited. When the discharge is profuse you should keep off your feet as much as possible.

2. Do not use alcohol in any form, as it always prolongs the disease. Drink only milk, tea, coffee, and from six to eight glasses of water during the day.

3. Avoid all sexual relations until you have been pronounced cured by your physician, as the disease may be given to a woman even after the discharge has apparently ceased. When it is present you should avoid sexual excitement, as erections always aggravate the disease.

4. Always wash the hands after handling the parts. The discharge, if carried to the eyes, will cause blindness.

5. Sleep alone, and be sure that no one uses any of your toilet articles, particularly towels and wash cloths.

6. Never lend your syringe to any one, and as soon as you are well destroy it.

7. Be sure that the bowels move every day. If they are inclined to be constipated, take a laxative.

8. Do not use mustard, pepper, horseradish, or stimulating sauces on your food. Do not drink ginger ale, beer, whiskey, or alcohol in any form.

LOCAL TREATMENT OF ACUTE GONORRHEA

The preventive and abortive treatment for acute gonorrhea have already been mentioned. The repressive treatment consists in the employment of local treatment calculated to control the inflammation, but with the prime object of lessening the symptoms, the complications, and the probability of chronicity and of aborting the acute attack.

Repressive treatment, if properly conducted, aborts the disease quite as certainly as the so-called abortive treatments. But inasmuch as it is founded on the theory that the urethra should not be irritated in the effort to slay the gonococci, its results, when it fails to abort, are far more satisfactory than those of the violent so-called abortive measures.

Cases Suitable to Repressive Treatment

The physician unfamiliar with the local treatment of urethral disease can expect but little success with the repressive treatment of gonorrhea. The expectant treatment will give him better results.

The physician moderately familiar with the subject should undertake this treatment with fear and trembling. He should apply it at first only to cases that he can absolutely control, who apply for treatment during the initial stage of the disease, before the meatus is much swollen, the discharge frankly purulent, the deeper portions of the urethra infected, or the pain on urination or erection at all marked. This admits most cases from one to three days old.

The expert will determine how far his personal success permits him to disregard the above rules.

In the clinic our control of patients is so poor that it is safer to employ repressive injections only upon patients who have had gonorrhea before and are peculiarly docile.

Acute gonorrhea in full blast, with marked swelling of the meatus, purulent discharge, and painful urination, is usually only made worse by local treatment. Exceptionally, however, and especially if the acute urethritis has persisted for a week or more, and a certain degree of local immunity has thus been acquired, very gentle injections, conducted exclusively by the physician himself, may control the acute inflammation.

Choice of Repressive Treatment

There are at present two schools of urologists: the one depends upon injection of organic silver salts for the repression of acute gonorrhea; the other upon irrigations with potassium permanganate. The success of the treatment with silver salts depends upon the painless and frequent injection of relatively strong antiseptic solutions in small[1] quantities.

The success of the permanganate treatment, on the other hand, depends upon the less frequent application of large quantities of a dilute solution. So that in the one method of treatment we attempt to destroy bacteria; in the other we attempt to wash them out. I used the permanganate irrigation treatment for years, but have found that, although it promptly controls the discharge, it often leaves the patient with a chronic urethritis, extremely slow to get well. The silver salts, although they do not control the urethritis so quickly, cure it in my hands far more rapidly.

Organic Silver Salts

For several years I employed argyrol almost exclusively in 10 per cent. solutions. But latterly I have found that protargol in 0.5 per cent. solutions is, if anything, more efficient, and infinitely cleaner. But the protargol is much more irritating than argyrol, and hence it often cannot be used at the outset. In such cases I employ argyrol until the irritation has subsided and then pass to the protargol.

The routine treatment is as follows: the patient with a beginning urethritis is examined in the first place to determine the presence or absence of gonococcus, and in the second place to determine whether the urethra is so inflamed as to prohibit repressive injection.

If injections are to be used the patient is placed upon the table, the end of the penis mopped clean, and the argyrol or protargol solution gently injected from a blunt-nozzle glass syringe until back pressure or a leak between the syringe and meatus indicates that the urethra is full. The syringe is then removed and the injection retained by gentle lateral pressure upon the lips of the meatus. At the first injection the urethra will usually not hold more than 1 dram (4.0 c. c.); later it will hold 2 (8.0 c. c.). As soon as the syringe is removed the patient is asked whether the injection pains. If so, the injected fluid is permitted to escape as soon as this pain increases. If not, as soon as the burning begins. The patient usually complains of increasing pain within a minute or so at the first injection.

If protargol has been used the patient complains of much more pain for the first 2 or 3 minutes after the injection than while this is being given.

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[1] Laboratory reports showing the relative inefficiency of the organic silver salts as an antiseptic are frequently published, and although they show that a relatively slight bacterial action is exerted by these drugs, it is nevertheless the universal testimony of those who treat acute catarrhal inflammation in various parts of the body that these organic silver salts are undoubtedly efficacious in controlling acute inflammation of mucous membranes. Thus argyrol, confessedly the weakest of them, is successfully used in the treatment of urethritis, tonsillitis, rhinitis, and conjunctivitis.

The patient is now instructed how to inject gently, holding the meatus open to receive the nozzle of the syringe, pressing lightly with thumb and forefinger on the lateral lips of the meatus. He is especially cautioned not to squeeze the meatus too tightly, but to retain the fluid by skill and not by force.

He is also instructed not to impede the inflow of the fluid by making pressure against the perineum, nor to encourage it by massage of the pendulous urethra, for massage of the acutely inflamed urethra is only calculated to do harm, while any effort to prevent the fluid from reaching the deeper portions of the urethra only keeps it out of the bulb, where it is most needed (after the first day or two). Indeed, the physician often finds it necessary to argue against the patient's fear of driving the gonococci into the deep urethra and bladder by injections. We well know that gonococci reach the deep urethra in at least 80 per cent. of cases not treated by injections, and it is evident to any one using injections that they do not actually drive gonococci ahead of them, but only encourage the advance of the gonococci, if they are used in such strength or with such brutality as to congest the urethra. For this reason the patient is especially instructed to avoid pain in all his manipulations. Inasmuch as pain is a relative term with a different meaning for each of us he is instructed to make all subsequent injections so brief and so gentle that the pain they excite shall be less than that excited by the first injection.

If the patient is intelligent and has given himself injections during previous gonorrheas, he may be dismissed with the instruction to repeat the injection 3 or 4 times a day, retaining the fluid in the urethra until he feels pain, or for 5 minutes. But again and again he must be instructed that, as soon as the injection begins to hurt, it is to be permitted to escape.

The patient reports to the physician for one of his daily injections until the latter is satisfied that the infection is fairly well controlled; as shown by the disappearance of the swelling at the meatus and the diminishing discharge. After this the patient may be permitted greater latitude, and may return for observation only every third or fourth day. In a promising case the discharge disappears completely in from 2 to 4 days, and the urine which the patient passes in 2 glasses at each visit shows only a slight haze in the first glass.

In the second week of the disease the discharge is likely to increase, and the problem now is whether this increase in discharge is due to the infection or to injection. In either case it is best to stop all injection for 24 hours, and meanwhile to examine the discharge carefully by smear for gonococci (there is no time for culture). Even if these are found it will usually be noticed that a 24 hours' remission of injection has resulted in lessening the discharge, and the injections are now resumed as before.

But, if no gonococci can be found in the discharge, the patient reports daily for examination, and, if necessary, he is given glass slides on which to smear his morning discharge. If 3 or 4 examinations fail to show gonococci, and the urine is becoming clearer all the time, the treatment is stopped, and the patient told to watch carefully for any sign of discharge, and if this appears he promptly reports for further examination. At the end of a week, without discharge or pus in the urine, he is probably cured, but should be seen a week later to be sure that the urine remains clean. If so, the cure is verified by submitting the first urine passed, as well as the urine passed after massage of the prostate and vesicles, to the test of culture.

VARIATION IN THE TREATMENT

No two cases can be treated precisely alike. The following chief variations in the treatment may be noted:

1. If the patient is first seen after his gonorrhea is well under way, but his physician still hopes to control it by repressive injections, he should use a mild solution (5 per cent. argyrol, 0.25 per cent. protargol) with the utmost gentleness and should closely supervise the progress of the case.
2. If pain supervene, either in the shape of increasing sensitiveness to injection, or pain on urination, or painful erections, the injection must be discontinued at least until these pains cease. If the pains do not cease upon the discontinuance of the injection, these may not be recommended, but the patient must be treated expectantly.
3. If the discharge continues during the 2 weeks in some quantity, or if pus appears in the second glass there is evidently posterior urethritis, and this must be attacked.

TREATMENT OF ACUTE POSTERIOR URETHRITIS

Acute posterior urethritis, like anterior urethritis, is to be treated by injection from the onset, if it is seen early. But if the posterior urethritis is sufficiently severe to cause painful urination, all injection must be stopped, and the disease treated by internal medication until this inflammation shall spontaneously subside. For the local treatment of mild beginning posterior urethritis I usually employ instillation of 0.25 per cent. protargol or of 1:10,000 silver nitrate once daily. If the patient is trained to take forced injections these solutions may be introduced with urethral instruments. Throughout the course of treatment frequent examination of the prostate should be made for fear of beginning prostatic abscess, and if the local treatment fails to control the local inflammation it must be stopped and the treatment of severe posterior urethritis be instituted (q. v.).

Permanganate Irrigation

The permanganate irrigation treatment of acute gonorrhea I have found less efficacious than the method just described. The method employed by Janet, who devised this system, is the following:

He irrigates the anterior urethra twice a day for three or four days, then increases the interval from twelve to eighteen hours. When the cloudiness of the first urine is almost gone he increases the interval to twenty-four hours. When the discharge is no longer purulent he makes it forty-eight hours.

When the second urine becomes cloudy he irrigates the posterior urethra according to the same method, twice a day at first, later every day, or every other day. For each irrigation of anterior or posterior urethra he employs 500 c. c. of fluid, at a temperature of 110° F.

If the case is seen before the appearance of marked inflammatory symptoms he employs a pint of 1:500 solution of permanganate, immediately followed by a like quantity of boric acid solution. If this does not prove too irritating he continues at this strength until the inflammation has subsided sufficiently to permit intervals of thirty-six to forty-eight hours, when he drops to 1:4,000 or 1:6,000 permanganate and omits the boric acid.

If the posterior urethra becomes inflamed he begins irrigating it with solutions of 1:4,000 down to 1:10,000. If these are well borne, he increases the strength to 1:2,000 or 1:1,000, and follows it with a boric acid irrigation.

If the patient is first seen after the appearance of acute inflammatory symptoms the irrigation is begun at 1:10,000 to 1:4,000 strength, and only for the anterior, even if the posterior urethra is inflamed. He begins treatment of the posterior urethra only when the anterior inflammation is under control.

In the declining stage he gives a daily wash of 1:6,000 to 1:8,000.

Other Methods

Valentine and the other followers of the Janet method in this country follow his method with certain variations. They usually employ much weaker solutions (1:4,000 to 1:20,000), and larger quantities (1,000 c. c. or more), and often irrigate the posterior urethra every day as a routine measure.

Expectant Treatment

The expectant treatment of acute gonorrhea consists in treating the disease solely by hygiene and internal medication, employing no local repressive measures, and beginning injections in the third or the fourth week, when the acute symptoms have begun to abate. Expectant treatment must be employed in all cases that are judged too severe when first seen for repressive measures, or prove rebellious to these. In some cases the treatment may be begun while discharge and pain are still quite severe, if one has waited in vain beyond the third week for any diminution of these. But as a rule it is safer not to inject until the local symptoms are decidedly diminishing.

Local Treatment of the Anterior Urethra.—If gonococci are still present in the discharge, the treatment is begun by anterior injection of protargol, as though the case were one of beginning gonorrhea. If this fails to do much good, after a few days permanganate irrigation is substituted: a pint of 1:6,000 permanganate solution, at a temperature of about 100° to 105° F. This solution is applied from the irrigator, the urethra being gently distended with the irrigator at a height of about two feet above the meatus. As soon as the anterior urethritis begins to improve, as shown by the lessening of the discharge, the posterior urethra requires treatment.

Local Treatment of the Posterior Urethra.—No treatment of the posterior urethra should be attempted until the inflamed anterior urethra is controlled. There is but one exception to this rule, mentioned elsewhere. The treatment of the posterior urethra is conducted by irrigation, as described under the treatment of chronic posterior urethritis.

TREATMENT OF COMPLICATIONS

Abscess of the Urethral Glands.—Periurethral abscess at the frenum or in the pendulous portion of the urethra is no contraindication to repressive injections, if these are conducted gently. The best treatment of these conditions is to leave them undisturbed until the abscess points, when it may be permitted to break internally, or may be incised externally.

Incision should be delayed until extreme suppuration is seen to be inevitable, for fistulæ are likely to result.

Periurethral Abscess.—Abscess in the glands of Cowper, or in the glands of the scrotal and perineal portions of the anterior urethra, usually spreads extensively beneath the skin before it suppurates frankly. It should therefore be incised as soon as a tendency to spreading is manifest. Meanwhile the repressive injections may be continued, unless the urethritis contraindicates.

Balanoposthitis.—Gonorrheal balanoposthitis can usually be prevented by cleanliness, and, if it occurs, is usually controlled by application of any nonirritating disinfectant powder. If the balanoposthitis is severe, the best wash is hydrogen peroxid, diluted to one-quarter strength with warm water. This should be applied twice a day, and the surfaces kept separated by a perforated piece of gauze.

Lymphangitis and Adenitis.—If the lymphatics of the dorsum of the penis become inflamed during a gonorrhea this is due to infection in the periurethral tissues or within the preputial cavity. When these are controlled the lymphatic inflammation subsides. The complication is rarely of any importance, for it is most unusual that it should go on to suppuration, requiring incision.

Gonorrheal inguinal adenitis is due to the same causes and rarely suppurates.

Acute Posterior Urethritis.—It is necessary to distinguish precisely those cases in which there is probably acute prostatitis from those in which there is none. If the prostate is not inflamed, symptoms of acute posterior urethritis call for cessation of all local treatment. The patient should not exercise at all, and it is preferable that he stay at home, if that is possible, while vigorous treatment is conducted with alkalies and sedatives, and hot rectal douches. If after a few days the symptoms continue very intense, in spite of this treatment, relief may perhaps be afforded by instillation into the posterior urethra of 2 or 3 drops of 2 per cent. silver nitrate solution. If pain persists in spite of instillation, the case is to be treated as one of acute prostatitis.

Acute Prostatitis and Prostatic Abscesses.—The best treatment of prostatic abscess is its prevention by the exercise of the hygienic precaution and the gentleness in local treatment already insisted upon. If abscess occur the treatment consists in:

1. Stopping all urethral treatment.
2. The administration by mouth of some soothing urinary antiseptic, with whatever sedative and laxative may be necessary.
3. Insistence upon the general rules of antigonorrheic treatment, especially as to physical rest (rest in bed, if there is fever).
4. Hot sitz baths or hot rectal douches, with the hot-water bag as a local sedative.
5. Catheterism and bladder wash if there is complete retention.

As a result of this treatment we look for prompt relief of two symptoms, viz., fever and retention.

If the patient's temperature does not, within a few days, fall to and remain below 100° F., and if acute complete retention is not almost immediately relieved, the abscess should be promptly operated upon.

Seminal Vesiculitis.—Inflammation of the seminal vesicules, during an acute gonorrhea, is overshadowed by the concomitant prostatic inflammation. Abscesses of the vesicle requiring incision are extremely rare.

Cystitis.—The treatment of gonorrheal cystitis is that of posterior urethritis as described above.

Pyelonephritis.—Pyelonephritis resulting from gonorrheal infection requires the same treatment as does that lesion when caused by other bacteria.

TREATMENT OF GONORRHEAL EPIDIDYMITIS

Prophylaxis

Gentleness and discretion in the treatment of acute gonorrhea are requisite for the prevention of epididymitis, yet one may expect to see a small percentage of cases complicated by inflammation of the epididymis in spite of every care.

The wearing of a suspensory bandage minimizes the danger of epididymitis, yet in many secondary gonorrheas one may dispense with this precaution. But during the first gonorrhea, and during the acute stages of posterior urethral infection, the suspensory should always be worn.

Posture

By far the most important element in the successful treatment of acute gonorrheal epididymitis is elevation of the testicles. If the attack is a mild one it may sometimes be aborted by proper support without putting the patient to bed or without applying any other treatment, yet such cases are very prone to relapse. When the inflammation is at all severe the pain is so intense that the patient practically has to remain in bed whether he wishes to or not.

While in bed the testicles should not only be elevated but elevated as high as possible. The ordinary strap of adhesive plaster across the thighs is quite inadequate for this work. The best bandage is Alexander's modification of Curling's bandage. This consists of a four-fold strip of gauze some 4 inches wide and 2 feet long. To the center of this is sewed the center of a piece of canton flannel 48 inches long in such a way that this canton flannel forms two separate tails to a "T" bandage. Another piece of canton flannel 3 inches wide and long enough to go about the waist of the patient is pinned in place as a supporting bandage.

A band of gauze is loosely folded into each groin and under the scrotum in such a way as to prevent the bandage from pressing too sharply against the skin, while at the same time elevating the testicles. The center of the "T" bandage is then applied in the perineum, the two ends of the folded gauze being laid along the groin, while the two pieces of canton flannel are carried back in the gluteal folds and pinned to the sides of the belly band. The folded gauze is then carried snugly up about the scrotum and penis to be fastened with a safety pin as closely as possible above the root of the latter, care being taken, however, that the end of the pin does not press into the skin. In this way the genitals are completely secured by a loop of gauze and are then suspended to the band by further safety pins. Small slips of gauze are then stuffed into all spaces about the scrotum, filling out all hollow places, and being pinned snugly, but not tightly, across the space left open in front of the folded gauze strip.

If the bandage has been properly applied, its lower surface stands at right angles with the long axis of the patient's body, and when so applied this bandage is calculated in almost all cases to dissipate the pain and fever within 48 hours. After the patient's temperature has been normal for 24 hours the parts are shaved and the bandage is replaced by adhesive plaster, narrow straps being run from the perineum to the hypogastrium, overlapping each other from the groin toward the median line and supporting the inflamed organ upon the belly. The only advantage of this adhesive plaster over the gauze is that, once in place, it cannot slip. This dressing is worn for at least two or three days while the patient goes about, until the sensitiveness shall have almost subsided. Then it is replaced by a suspensory bandage or "jock-strap."

A less efficient support, while the patient is in bed, is the ordinary "jock-strap," or a "T" bandage, made with a band and a towel.

General Treatment

The bowels are likely to be constipated, and this must be attended to. The patient is kept upon a fluid diet as long as the temperature is above a hundred. Sometimes the administration of one or two minims of tincture of aconite every two hours is efficacious in relieving pain, by dilating the blood vessels. The other remedies employed, such as gelsemium and veratrum viride, are doubtless no more efficacious.

Vaccine Treatment.—After some years of experimentation I am still in doubt as to the value of vaccines. In my private practice, where the patients are not fully under control and the treatment by rest and suspension, therefore, not so efficacious, I usually administer 50,000,000 gonococci every two days for three or four doses. In my hospital practice, however, the patients are so promptly relieved by elevation of the testicle that there seems to be no advantage in the use of vaccines.

Local Application.—A great number of local applications have been employed at various times in the treatment of this inflammation. One's preference is largely a matter of fashion. Thus the tobacco poultice once universally used is now scarcely mentioned. Its virtues consisted in its heat, and one obtains this as well with the more familiar flaxseed poultice, somewhat less well with a hot-water bag. Strong theoretical objections have been urged against the use of cold, yet many patients obtain much more relief from the application of ice bags than from poultices, and I usually apply an ice bag over the bandage while the patient is in bed, unless the pain is promptly relieved by the bandage alone. Applications of pure guaiacol or equal parts of guaiacol and glycerin are painful, but sometimes appear to help on the first day of the disease. I used to employ them constantly but have given them up of late years. The saturated solution of Epsom salts I have not found efficacious.

Treatment of the Declining Stages.—Most cases require nothing more than good suspension after they have left bed, but if the edema is slow to disappear resolution may be hastened by strapping.

First tie a soft cotton string tightly around the scrotum above the inflamed testicle so that this cannot slip upward. Then take a piece of thin rubber bandage some two inches wide and long enough to surround the swollen gland; apply to the end of this a short narrow strip of adhesive plaster, and strap the rubber bandage as tightly around the testicle as the patient can bear it, fastening it in place with the adhesive plaster so that it shall not slip off. The advantage of this form of strapping is that it does not adhere inconveniently to the skin and may be reapplied as tightly as the patient can stand it every day, or every other day, as the swelling recedes.

Treatment of the Urethra.—No local treatment of the urethra is permissible during the attack of epididymitis, nor for a week, or even more, thereafter, lest irritation of the urethra should excite relapse.

Operative Treatment.—The observations of Hagner have stimulated interest in the operative treatment of epididymitis. Although I have operated upon a number of cases, I do not feel that the knife should be employed excepting in those rare instances when proper support, the application of cold or heat (whichever is more grateful), and aconite fail to relieve pain. Moreover, operation can only be undertaken with the knowledge that the 5 days required for the healing of the wound, under the best of circumstances, may keep the patient rather longer in bed than if he had not been operated upon.

Treatment of Recurrent Epididymitis.—Immediate recurrence is usually prevented by abstention from local treatment of the urethra. On the other hand a tendency to relapsing epididymitis may often be controlled by prostatic massage, while in certain cases relapse appears to depend upon a small smoldering focus of suppuration in the epididymis. This may be attacked either by vasotomy or by incising the little mass in the epididymis.

CHRONIC GONORRHEAL URETHRITIS

General Treatment

It is difficult to define precisely what should be the general treatment of chronic gonorrheal urethritis, for this treatment, beginning at the termination of the acute stage, takes up the patient at a time when exercise and alcohol are absolutely prohibited with the object of carrying him from that condition into one of free exercise and sometimes of free alcohol, neither putting him back by rashness nor delaying him by overcaution. It is not sufficiently recognized by most medical men that there comes a time when a patient with gonorrhea, who has been prohibited from exercise for a number of weeks, goes "stale," as the athletes say, and the young man thus deprived of his accustomed exercise becomes morbidly depressed, so that he can scarcely be expected to throw off his infection. Under these circumstances, even though gonococci persist in the urethral discharge, a patient must be advised to begin exercise, at first very gently.

The best beginning is made with dumbbells or some similar form of exercise that puts most of the strain upon the arms. Vigorous leg work, such as tennis and swimming, should not be attempted until the patient has had at least a week or two of preliminary experiment of a milder sort.

Thus feeling his way, the physician attempts not only to get his patient back to a normal manner of living, but even to make him exercise rather more than is his custom. The mental and physical stimulation of this often goes far to cure an intractable chronic case. On the other hand, the possibility of setting up acute infection in the urethra, the prostate, or the epididymis, by intemperate exercise or straining, is never to be forgotten. This danger sometimes persists even after gonococci have disappeared.

A similar difficulty exists in the regulation of alcohol. Alcohol makes the urine irritating to the urethra, and such irritation is inevitably bad for acute urethritis. But after the gonococci have disappeared (and sometimes even before this) the irritation of alcohol may (exceptionally) be beneficial rather than harmful, for it is to be remembered that some of the drugs that we apply in the local treatment of chronic urethritis act chiefly as counterirritants. Thus the alcoholic, who boasts that, after months of chronic gonorrhea, he has thrown physics to the dogs and gone on a wild debauch, which has cured him, often speaks the truth. Happily, however, the physician need not employ such intensive treatment. One or two drinks of beer a day will do as much good (and far less harm) as many times that number. One must remember, however, that alcohol is, in the majority of cases, and unto the bitter end, much more harmful than beneficial.

Medication.—The various alkalies and balsamics, which are so useful in acute urethritis, are usually of no benefit in chronic cases, except during acute relapses. If the urine is so acid as to be constantly full of irritating crystals, this tendency must be corrected. On the other hand, some benefit may occasionally be derived from free water drinking, and it is not to be forgotten that some cases of chronic gonorrhea depend for their continuance upon some other lesion not connected with this malady, such as tuberculosis, nephritis, or diabetes.

Change of Surroundings.—When exercise and hygiene and medication all fail to bring a patient mentally and physically up to par, and he continues to drag on weary months of chronic urethral catarrh in spite of intelligent local treatment, he may sometimes be cured by sending him away for a vacation. It matters not where he be sent if only the locality be healthy, his occupation and method of living be radically changed, and his tastes be consulted. One has recourse to this method of treatment rarely, yet I have seen it followed by the best of results, the patient getting well either during his trip or immediately upon his return.

Sexual Hygiene.—While gonococci persist, sexual intercourse is as likely to reinfect the gonorrheic as it is to infect his partner. But, after their disappearance, it is likely to do good by relieving the sexual congestion of one who is (presumably) accustomed to frequent sexual intercourse. The irritation of ungratified sexual desire, the effort to check the sexual habit, is to many gonorrheics the most distressing feature of the disease.

Local Treatment

No absolute rules for the local treatment of chronic urethritis can be made to apply to all cases, for the condition consists of a chronic catarrh, which, while it chiefly attacks the prostate, the seminal vesicles, and the surface of the posterior urethra, also involves the anterior urethra, or may be confined in the latter portion of the canal, and which may or may not be complicated by stricture.

A precise diagnosis of the site, character, and obstinacy of the lesions, as well as the presence or absence of gonococci, is an essential preliminary to intelligent local treatment.

Thus we have cases that will get well rapidly if let alone, others that will never be cured unless they are treated locally, others again peculiarly susceptible to certain forms of treatment and made worse instead of better by measures that one would suppose, *a priori,* calculated to do good. Nor are we speaking of exceptional cases, unless indeed we might say that every case of chronic urethral gonorrhea is an exceptional case. To the beginner this is so, no matter what rules he may follow. He cannot be too careful in applying them, and must attack each urethra for the first time, whether by injections, irrigations, sounds, or urethroscope, with a great fear in his heart. The expert knows no rules, and is a misleading guide, because he inevitably presupposes in the beginner some of the discretion and dexterity which he has unconsciously attained through years of experience and practice.

Hence, whatever may be hereinafter set down must be accepted with reserve, and applied in a purely experimental way, for each practitioner must learn through his own experience in how much all rules are applicable to him, and in what measure his hand and mind may employ them to his patients' advantage.

Injections.—The first local treatment to be employed upon a patient with chronic urethritis is injection into the anterior urethra. This injection is made by means of a 2-drachm (8.00-c. c.) blunt-nozzle piston syringe. It is immaterial whether the syringe be made of glass or hard rubber, and whether or not it have a soft rubber tip, so long as its point is blunt and its piston tight.

A great variety of solutions are employed, but it is the general rule that the organic silver solutions, of which protargol in 0.25 to 0.50 per cent. solution is the type, are usually the most efficacious remedies when the discharge is profuse and creamy and full of gonococci. Yet even in these cases these organic silver solutions are sometimes useless, and the so-called astringents are preferable. In all milder cases, if there is a discharge through the day, astringents are likely to help control this. The most favored injections have as a foundation some of the forms of zinc.

Zinc sulphate, in 0.2 per cent. to 1 per cent. solutions, is frequently used.

Potassium permanganate, in 1 to 3,000 to 1 to 5,000 strength, is, I think, usually of little value.

Zinc permanganate (1:2,000) I think well of.

My favorite injection is:

℞ Zinci sulphatis 0.25 gr. iv
Liq. plumbi subacetatis diluti, 100.00 ℥iii
M.
Sig.—Shake. Inject morning and night.

The astringents favored by other authors are:

℞ Hydrargyri chloridi corrosivi, (0.03) gr. ss.
Acidi carbolici, (0.8) m. xii.
Zinci sulphocarbolatis, (0.8 to 4.0) gr. xii to ʒj.
Boroglycerini (25 per cent.), (60.0) ℥ij.
Aquæ, q. s. ad (200.0) ℥vj.
(White and Martin).

℞ Zinci acetatis,
Acidi Tannici, āā (1.3) gr. xx.
Aquæ rosæ, (125.0) ℥jv.
(Ibid.).

℞ Zinci sulphatis, (1.0) gr. xv.
Plumbi acetatis, (1.3) gr. xx.
Tincturæ opii; Tincturæ catechu, āā (65.0) ʒij.
Aquæ, (200.0) ad ℥vj.
(Brou).

℞ Zinci sulphatis; Aluminis, āā (0.3 to 0.6) gr. jv. ad gr. xij.
Acidi carbolici, (0.3) m. jv.
Aquæ, (125.0) ℥jv.
(Ultzmann).

℞ Zinci sulphatis, (0.7) gr. xij.
Resorcin, (1.5) gr. xxjv.
Aquæ, (125.0) ℥jv.
(Morton).

℞ Acidi nitrici, (0.18 to 1.0) M iij to xv.
Aquæ, (250.00) ℥viij.
(Baumann).

℞ Cupri sulphatis, (0.2) gr. iij.
Aluminis crudi, (1.0) gr. xv.
Aquæ, (250.00) ℥viij.
(Kreissl).

℞ Extracti hydrast. fl.; Bismuthi subcarbonatis; Boroglycerini (25 per cent.), āā (25.00) ʒvj.
Aquæ destillatæ, ad (200.00) ℥vj.
(White and Martin).

℞ Ichthyol, (1.3 to 6.0) gr. xx to ʒjss.
Aquæ, q. s. ad (125.00) ℥jv.
(Baumann).

℞ Berberine hydrochlorat., (0.3) gr. v.
Aquæ, (250.00) ℥viij.
(Belfield).

It were vain to try this formidable array of drugs upon any one patient. If one or two of them fail, the rest are likely to, for the failure is more often due to the inaptitude of the method of treatment than to inappropriateness of a given drug.

Excepting the organic silver salts, none of these injections should be retained more than long enough to distend the canal. It is unwise to employ them oftener than twice a day, or less often than once a day.

Irrigations.—The therapeutic effect of injections into the anterior urethra is obviously restricted to that part of the canal, though gonorrheal inflammation almost inevitably extends to the posterior urethra. Hence, injections are useful in bringing the anterior urethra to a condition to permit the passage of instruments and fluids necessary for the treatment of posterior urethritis, and also for the purpose of calming the patient's apprehension by making him feel that he is doing something for his discharge; but the real work must usually be done with irrigations or instillations to reach the posterior urethra.

CHOICE OF METHOD OF IRRIGATION.—The urethra may be irrigated with or without a catheter. Irrigation without a catheter is preferred in many clinics, because it is more readily performed and is adapted to most cases.

But, though there is doubtless no material difference in the therapeutic value of one or the other method, if properly employed, the routine employment of irrigation without a catheter permits the physician to overlook many lesions, such as stricture, retention of urine, etc., which would be called to his attention if he followed the other method. I therefore much prefer, and employ, the catheter.

TECHNIQUE OF IRRIGATION WITHOUT A CATHETER.—The instruments required are a glass nozzle to fit the meatus and an irrigator hung upon the wall in such a way that it may be elevated or depressed at will. The patient first urinates, then stands over a sink (though the first injection under these circumstances may result in his fainting, and it is, therefore, preferable that the patient lie down until he becomes accustomed to the treatment). The irrigator is filled with the solution to be injected, elevated 3 to 4 feet above the urethra, and the fluid then permitted to enter the urethra gradually. As the patient feels pressure upon the wall of the canal he is instructed to relax his muscles by going through the motion of urinating without endeavoring forcibly to expel the urine. Other patients manage better if simply told to relax their muscles, while others again do better if told to breathe in and out rapidly, imitating the panting of a dog. Some patients readily learn the trick, others defy all efforts to train them. One obtains better results with some by employing a 4-ounce piston syringe, with a rubber nozzle coupled to it; with this the pressure can be varied in proportion to the resistance of the urethra.

TECHNIQUE OF IRRIGATION WITH CATHETER.—The entrance of instruments into the urethra should always be cleanly and gentle.

ASEPSIS.—The asepsis of catheterism implies three requisites:

1. Asepsis of the physician's hands.
2. Antisepsis of the patient's urethra. And
3. Asepsis of the instrument introduced.

The best rule of cleanliness for the physician's hands is that, having washed his hands, he should act as if they were still dirty. The last three inches of the instrument should not be touched by anything except sterilized lubricant from the time it is sterilized until it enters the urethra.

Asepsis of the patient's urethra is, under the present circumstances, amply cared for by the antiseptic of the solution, which is to be introduced through the catheter, and which will wash the urethra clean upon its emission.

Asepsis of the Instrument.—This implies three conditions:

Aseptic lubrication.

Cleanliness and sterilization after use.

Aseptic preservation.

Lubrication.—Oily lubricants, such as vaselin or olive oil, though they themselves may be sterilized by boiling, prevent proper sterilization of the instrument to which they are applied by coating it with an imperceptible oily film very difficult to remove. Thus Albarran found that, while an unoiled catheter may be cleansed by boiling for ten minutes, an oily catheter required thirty minutes' boiling to clean it. Moreover an oily lubricant cannot be employed for cystoscopes. Glycerin and boroglycerid are frequently employed. The following modification, by Doctor Ruggles, of Casper's formula is the one I employ:

Dissolve 1 cgm. of oxycyanid of mercury in 200 c. c. of hot sterile water; add 35 c. c. of glycerin, and water enough to make 350 c. c. Let this mixture cool; then add 10 to 15 gm. of powdered gum tragacanth. Let this stand until it becomes a homogeneous mass, a process which takes several days, but may be hastened by occasional stirring to break up the lumps. The amount of tragacanth employed depends upon the consistence of this substance, which varies considerably.

This lubricant may be put up in sterile paint tubes. Its quality depends upon the employment of precisely the right amount of tragacanth.

Instrument Sterilization.—All urethral instruments should be sterilized immediately after use. Sounds and rubber catheters may be readily sterilized by boiling. Woven instruments of the best French makes may also be boiled, if the following rules are observed:

The boiler must be long enough to contain the instrument without bending it, and without touching it at either extremity. The instrument must be completely immersed in the water. The instrument must not be bent in the least degree before it has cooled. Therefore it is best to boil it in a receptacle with a tray upon which the instruments are lifted out and cooled somewhat before they are used. Finally the instrument should not be boiled for more than 15 minutes at a time.

Cystoscopes cannot be boiled. They must be sterilized in formalin vapor. No peculiar apparatus is required for this. The instrument must simply be kept in an air-tight instrument case, in which a formalin lamp is lighted, for at least an hour.

Needless to state, all instruments should be mechanically cleansed with soap and water and dried before they are sterilized.

Aseptic Preservation of Instruments.—The instrument case to contain catheters, sounds, etc., may be sterilized with the formalin lamp, and it is convenient to have the sterilized instruments thus preserved in a sterile case. Yet one must observe the precaution of dipping them in sterile water before using them lest the deposit of formalin upon them irritate the urethra.

Asepsis of Tanks, Syringes, Etc.—It is my custom to keep all small syringes, hypodermic needles, mixing rods, and instillators in a 20 per cent. formalin solution, supersaturated with borax.

Large syringes may be sterilized with formalin in the instrument case.

Wall tanks, graduates, etc., may be kept sterile by using them only for antiseptic solutions. It should be the urologist's practice to have a special type of glass for urinary specimens, in order that these may not by any chance defile his solution containers.

Asepsis of Solutions.—All solutions should be made up fresh, warm, and aseptic, a supply of hot, sterile water being at hand. The chemicals to be kept in stock for solution are shown in the following table, which also indicates the strength in which they are commonly used:

Name.	Form.	Injection.	Irrigation.	Instillation.
Argyrol	Crystals	5-20%[1]	3 -10%	10–50%
Protargol	0.5 gm. powders	0.25–1%[1]	0.1–0.5%	1–5%
Albargin	0.2 gm. tablets	0.1–1%[1]	0.05–0.2%	1–5%
Potass. permang	1 gr. tablets	....	0.01–0.03%	....
Silver nitrate	10% sol	....	0.01–0.02%	0.2–10%
Hg. oxycyanid	0.25 gm. powders	....	0.03–0.2%	....
Zinc sulphate	1% sol	0.5–4%	0.1–0.5%	....
Zinc permang	1% sol	0.2–0. %	0.05–0.2%	....
Copper sulphate	10% sol	....	0.02–1%	0.5–5%

[1] Must be retained three to ten minutes in the urethra.

Instillations.—The term instillation is applied to the treatment of urethritis by the application to the urethra of a few drops of concentrated solution, while irrigations consist in the application of a large amount of relatively dilute solution. Instillations are usually applied only in the posterior urethra; application of strong solution to the anterior urethra being preferably made through the urethroscope.

Instruments Employed.—Although an instillation may be made with a soft-rubber catheter and a piston syringe, it is so difficult to gage the precise amount injected that it is preferable to use special instruments. Of the two instruments employed for this purpose, that of Guyon consists of a silk-woven capillary tube with a bulbous extremity, in which are one or more orifices and a funnel end for the adaptation to it of the nozzle of a syringe. The Keyes instillator consists of a small blunt metal catheter. with a capillary lumen, and the orifice at the tip, while the outer end is fitted with a screw to adapt it to a hypodermic syringe. Syringe and catheter are also made in one piece, but such an instrument is not convenient. The Guyon instrument has the advantage of passing more painlessly into the urethra, but it is not always possible to insert it into the posterior urethra, and the instrument is not durable.

The Keyes instrument is more difficult to pass painlessly, but it can always be inserted into the posterior urethra, and is much more durable.

Technique of Instillation.—The instillator is filled with the solution to be employed, and gently introduced into the urethra until its tip is in the membranous urethra. The contents of the syringe are then ejected into the membranous and prostatic portions of the canal. One can usually tell when the instrument has entered the membranous urethra by feeling its tip ride over the bulbo-membranous junction.

If the Keyes instillator is being used any doubt as to the position of its tip may be settled by noticing the position of the shaft of the instrument. So long as the point has not entered the deep urethra the shaft will tend to incline at an angle upward toward the patient's body. As the point engages, the shaft naturally falls downward toward the patient's feet. After withdrawing the instrument, if the fluid has been injected into the posterior urethra, none of it flows from the meatus.

Solutions Employed.—Silver nitrate, in strength of from 0.2 to 10 per cent., is the favorite solution for instillation. Copper sulphate is also employed in the same strength. Sublimate, 1:20,000 up to 1:2,000, is instilled in the treatment of tuberculosis, and thalline sulphate is employed for cases that are too sensitive for silver nitrate.

Instillations have an unmerited bad name among the laity, because it is so easy to instill into the posterior urethra an unduly strong solution of silver nitrate which will cause acute suffering for many hours. This is quite unnecessary, but can be avoided only by the exercise of the greatest conservativeness in making the first injection. The sensitiveness of the posterior urethra varies to a remarkable degree. Some patients are tortured beyond endurance by the instillation of 0.5 per cent. solution of silver nitrate, while others bear with composure an instillation of 10 per cent.

It is therefore wiser to test the sensitiveness of the posterior urethra by the passage of instruments and the use of irrigations before attempting instillation, and always to begin with a solution no stronger than 0.2 per cent. Moreover, inasmuch as the pain excited by instillation varies up to a certain point in proportion to the amount of fluid injected, it is better, at least in the beginning, to instill not more than two drops of the solution, and, inasmuch as it is often the object of this treatment to apply the strongest solution that the patient can bear, it is often better to instill only this minute dose in order that the strength may be more rapidly increased.

USES OF INSTILLATIONS.—Instillations are employed under three circumstances:

When the patient suffers from acute posterior urethritis, unaccompanied by a palpable change in the prostate and vesicles, but associated with persistent and intensely painful and frequent urination, the instillation of a few drops of 1 per cent. silver nitrate into the posterior urethra, though it may be extremely painful, is sometimes followed by the most remarkable relief of symptoms. If the first instillation does not help it should not be repeated.

When the posterior urethra has recently become inflamed and no instruments have previously been employed, less trauma is inflicted upon this portion of the canal by treating it with instillations than by any other form of treatment, provided the instrument is skillfully introduced. Under such circumstances one desires to apply rather large quantities of relatively dilute solutions, such as 10 c. c. of 0.1 per cent. silver nitrate, or 0.5 per cent. protargol, or 10 per cent. argyrol.

The routine employment of instillations, however, finds its place in the treatment of mild chronic posterior urethritis.

Here the dose is a few drops, the favorite remedy silver nitrate, beginning at 0.2 per cent. and increasing at intervals of twice a week. A similar technique is employed for instillation after the passage of sounds.

Dilatation and Massage.—Many chronic gonorrheas recover with no local treatment whatever, or under treatment of injection and irrigations. But, if there is urethral stricture or chronic prostatitis and vesiculitis, these lesions, though they sometimes cease to give symptoms under such treatment, often do not yield to it, and even when they do are likely to cause a relapse of symptoms after a shorter or longer interval.

The treatment of stricture is such a special subject that we must deal with this apart.

The treatment calculated to cure chronic anterior urethritis, prostatitis, and seminal vesiculitis is dilatation and massage.

The action of urethral dilatation and massage of the prostate and vesicles upon chronic intractable catarrh of these organs is twofold. In the first place the treatment actually expresses from the tissues the pus and bacteria within the urethral and prostatic glands. In the second place it softens inflammatory exudates and encourages a more firm and normal contraction of the urethral and prostatic muscles about the inflamed glands, at the same time producing hyperemia, which encourages the resorption of inflammatory tissue and the cure of glandular catarrh.

One might suppose, therefore, that every intractable urethritis required dilatation, but this is far from being the case. In certain patients the symptoms are only aggravated by these mechanical treatments, and, though temporary aggravation is not always a bad sign, yet if repeated gentle treatments continue to provoke an increase of symptoms, the mechanical violence is evidently doing more harm than good, and the patient is better without them. Hence, it is well to reserve massage, and even more carefully to reserve dilatation, for those cases that are incurable without it. One can scarcely be too enthusiastic about the advantage of these methods of treatment if one constantly bears in mind the possibility that they may do harm.

Technique of Dilatation.—As a general rule, if examination with the bulbous bougie or the urethroscope reveal an induration in the anterior urethra, which is not promptly ameliorated or cured by irrigations, it should be dilated. Dilatation should be begun with sounds, and these should be carried to the limit of the meatus; progress being made slowly, not more than two or three numbers at a given occasion. The passage of the sound should be preceded by the administration of hexamethylenamin, 7½ grains (0.5 gm.), t. i. d., for 48 hours, and followed by an instillation along the whole urethra of a few minims of 0.2 to 0.5 per cent. silver nitrate or by an irrigation with 1:5,000 potassium permanganate or 1:10,000 silver nitrate.

With the sound in the urethra this canal should be carefully palpated for infiltrations or glandular indurations, and these should be gently massaged upon the sound every time it is introduced until they disappear, or until it becomes evident that they are permanent scars.

The passage of the sound may be repeated as often as twice a week if it excites no increase in discharge. But, if the sound irritates, it should not be reinserted until this irritation has subsided, and, in such cases, it is preferable not to repeat the sounding oftener than once in 5 or 7 days.

When the limit of the meatus has been reached, dilatation should proceed by means of the Kollman dilator. Never having employed the irrigating dilator I cannot discuss its advantages. I have at times employed the various dilators that are adapted to distend only certain portions of the canal, selecting the instrument in accordance with the urinary and urethroscopic findings. But I very gravely mistrust the precise accuracy of these means of diagnosis, and, since the inflamed portion of the canal is always narrowed, I have come to believe that dilatation of uninflamed regions is not so likely to do harm as is the possible oversight due to dilating only one portion of the canal when actually the whole canal requires treatment. Therefore I now employ almost exclusively the Kollman dilator that stretches the whole canal.

Much more experience is required in employing the dilator than the sound, for, in introducing the sound, the resistance of the indurated portion of the canal or the bleeding which follows its removal are indices to guide the gentleness of the manipulator.

But, in using the dilator, the physician is working, not only against the resistance of the walls of the canal, but also against the resistance of the mechanism of the instrument itself, and therefore the amount of force justifiable in the use of one dilator is no guide to that justifiable with another. As a general rule, the dilator should be used so gently as to excite no bleeding and no inflammatory reaction for longer than 24 hours. The force required for this is different for each instrument and for each case. The increase in size with each instrumentation should be as in the use of the sound; not more than two or three numbers of the French Charrière scale.

There appears to be no advantage in leaving sounds or dilators in the urethra for more than a few moments after the desired dilatation has been achieved.

If the introduction of any metal instrument causes bleeding, small instruments (20 to 25 F.) should be repeatedly introduced until this tendency to bleeding has been overcome.

Contraindications to Dilatation.—Dilatation is always dangerous while gonococci still persist in the urethra, and, under these circumstances, the dilator is more dangerous than the sound. The same rule holds true so long as the urine contains free pus, even though no gonococci can be found. Yet sometimes gonococci and free pus cannot be gotten rid of excepting by dilatation.

On the other hand, when the urine contains only shreds, dilatation is more likely to do good, less likely to do harm.

Exceptionally, dilatation does harm when carried to the posterior urethra, though it is required in the anterior urethra.

The harm done by dilatation consists in exciting pain or an increase in the flow of pus from the urethra or in causing epididymitis.

Limits of Dilatation.—The natural limit to dilatation is the cure of the patient's symptoms, but certain restrictions may be put even upon this. When dilatation does good it should be carried to at least 28 or 30 F., and it is wise to see the patient a year later to be sure that no relapse of urethritis or contraction of a beginning stricture has taken place.

Some cases, on the other hand, are benefited by dilatation up to 35, 40, and 45 F. But many urethras resent being stretched to such dimensions, and dilatation should always be desisted from when it appears to do harm rather than good.

Techique of Massage.—Massage of the prostate and vesicles should be practiced with two principles in mind. In the first place, the whole of these organs should be massaged, even when the greater part of them may appear normal. In the second place, more attention should be paid to those regions that are palpably diseased.[1]

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[1] Diagnosis of inflammation of the prostate and seminal vesicles depends upon

The question whether it is better to massage prostate and vesicles gently or harshly is one that cannot be categorically answered. The more recent or the more acute the inflammation of the parts the milder should be the massage; and, in my belief, even old chronic cases do better under prolonged gentle massage than under more vigorous handling. But no one can tell how hard any other man massages. Indeed, it is probable that no one employs precisely the same amount of force in two successive treatments. Therefore this question must be left to the discretion of each individual, with the warning that severe massage is more likely to excite inflammatory reaction, although it is required in some intractable cases.

One cannot perform satisfactory massage with instruments. The sense of touch is necessary for delicate and accurate manipulation.

A simple method is to begin upon one vesicle, and, reaching up as far toward the fundus as possible, to press upon it, and then withdraw the finger in a zigzag way until one reaches the prostate. This maneuver is repeated half a dozen times, and then the same treatment given to the opposite vesicle. If the vesicles are impalpable, this is enough. If distended or indurated, the maneuver should be repeated often enough to make a distinct reduction in their size, if the patient can bear so much manipulation.

The finger is then brought down to the prostate. Hard, angular indurations in and about this organ had best be avoided, and pressure made chiefly upon the more yielding portions of the gland. Beginning with one lobe, pressure is made upon it either with a to-and-fro lateral sweep of the finger or with a circular motion. This manipulation, if gentle, may be continued for one minute; if severe, half a dozen strokes may suffice. The same treatment is given the opposite lobe of the gland, and the manipulation concluded by a half dozen strokes over the prostatic sinus for the purpose of emptying the main ducts into the urethra.

The progress of the cure is gauged by the amount of pus (as seen under the microscope) expressed from the meatus or passed in the urine after massage.

Irrigations are employed in connection with massage in order to wash away the pus extruded into the urethra and also to heal its superficial lesions. While gonococci are present, it is usually wiser to irrigate, but, after these have disappeared, one sometimes does better by omitting all intraurethral treatment.

Mild massage may be repeated twice, or, exceptionally, three times a week; severe massage not oftener than once a week. Massage every day almost invariably makes the patient worse.

Massage should be continued until the subjective symptoms are relieved, and the return shows no more than a few leukocytes to each micro-

the discovery, by means of the microscope, of pus in the secretion expressed from these organs. Hence in many cases we massage organs that are not palpably diseased.

scopical field. When this point has been reached, it is well to discontinue massage for a month or more, when the patient returns for another treatment. If he is doing well the pus is usually found to have decreased. If pus has reaccumulated, a few rubs usually bring it down again, and the patient should be continued under treatment in courses of four to six rubs and with intervals of two to four weeks until the reaccumulation of pus ceases.

Contraindications to Massage.—Massage is dangerous only in the presence of acute inflammation of the urethra, the prostate, the vesicle, or the epididymis; but massage is harmful in case it increases the patient's subjective symptoms instead of relieving them. It is also harmful in case it so hypnotizes the patient that he thinks he must come for the rest of his natural days to be rubbed for the relief of imaginary discomforts. Such patients should be discouraged from massage by all possible means. Their proper cure is sexual relief by matrimony.

The Rectal Douche.—The rectal douche is an accessory or substitute to massage of the prostate and vesicles. The usual case, that can perfectly well submit to massage, need not bother with douches. But if the patient cannot reach his physician often enough for massage, if the inflammation is too acute for massage, or if massage proves irritating or in any way harmful, the rectal douche should be employed. The object of the rectal douche is to apply heat or cold to the prostate or vesicles. For this purpose the closed tube, or psychrophore, may be employed, but the open double-current tube is better.

If no double-current tube is to be had, Tuttle's apparatus may be employed. It consists of two large soft-rubber catheters, bound or sewed together, side by side. The water flows in through one, out through the other. When the outlet is plugged with feces the current is reversed. Of the special tubes, I find Chetwood's model more convenient than those of Kemp or Tuttle.

The patient fills a two-quart douche bag with saline solution at 125° F., attaches it to the tube, hangs the bag so that its elevation above the outflow shall be about two feet, and greases the tube with vaselin.

He then seats himself toward the back of a privy seat, leans back against the wall, grasps the tube with his thumb at about its middle, opens the cut-off of the douche bag until the water flows warm through the tube, and then inserts the tube into the rectum for about half its length. He then turns the water on, and it flows into the rectum. If it does not return through the outflow he stops the inflow as soon as the rectum feels full, pokes about with the tube until a gush of water announces that it is in the right position, then turns the water on again. It takes from four to eight attempts before the patient learns to do the trick neatly.

The injection should be repeated every day, with an interval of a few days, every two or three weeks, to make sure that the bowel is not being irritated. Some patients note an immediate sense of relief from the use of this rectal douche, but the majority do not, and it is often difficult to persuade a patient to go on, week after week, using a treatment which is a great nuisance and which does not appear to him beneficial. Yet the rectal douche is one of the few forms of treatment that I consider it wise to continue for months at a time, with only such intervals as are necessary to insure the safety of the bowel.

Operative Treatment

Although Young and Alexander have counseled prostatectomy for the treatment of chronic prostatitis, an operation is likely to do more harm than good unless there is acute abscess, chronic obstruction in the form of urethral stricture, or bar or stricture at the neck of the bladder giving symptoms similar to those of prostatic hypertrophy.

Ligation of the vas deferens sometimes exercises a markedly beneficial effect upon intractable prostatitis and vesiculitis, but, since this operation makes the patient sterile, it is permissible only upon old men and upon such young men as suffer from relapsing epididymitis, incurable by any other means.

Urethroscopic Treatment

The urethroscope is more generally applicable to the diagnosis than to the treatment of urethritis. Intractable cases of urethritis may, however, be due to persistent suppuration in one or more single follicles or glands in the anterior urethra, or to suppuration in a paraurethral duct. In either case, direct treatment through the urethroscope is indicated. Injections of a drop of 20 per cent. silver nitrate solution may be made once or twice a week, or, if the canal is long, it may be slit up into the lumen of the urethra, whereupon it will promptly heal.

Such conditions are rare, however. As a rule, chronic urethritis is a diffuse process, although certain spots in the mucosa show more evidence of inflammation than others, and treatment of these spots by application to them of silver nitrate solution, introduced on a swab through the urethroscope, is not so likely to effect a cure as is the treatment of the sclerosis by dilatation.

Urethral polyps, granulations, or papillomata may be conveniently burned off through the urethroscope by means of the high-frequency current, the galvanocautery, or by repeated application of 20 per cent. silver nitrate solution.

If there are numerous urethral warts, it is convenient to destroy these in part by catching them between the edge of the urethroscopic tube and a large cotton swab, which is pushed beyond the warts and then withdrawn so as to amputate them against the edge of the tube. After the irritation from this procedure has subsided, the base is burned off.

The posterior urethroscopes of Goldschmidt and Buerger have materially helped in the diagnosis of the lesions of the posterior urethra, but their therapeutic efficiency has not yet been found to be very great. In the treatment of the verumontanum the Swinburne urethroscope is usually more serviceable.

Treatment of Postgonorrheal Urethritis

After gonococci have disappeared from the urethral and prostatic discharges, the sclerosis and glandular catarrh may persist for an indefinite time in the urethra or in the prostate or seminal vesicles. Under such conditions the treatment is much the same as that for chronic gonorrheal urethritis, with the exception that dilatation may be employed with more impunity and safely carried to a greater degree than when gonococci are still present.

One important thing to realize in these cases, however, is the necessity of stopping local treatment at a certain point. This point is usually reached when the secretion expressed from the prostatic contains only a few leukocytes; when there is no longer any free pus in the urine; when urinary shreds have become relatively small and contain very few pus cells; when the urethral discharge has become mucoid and sticky in character, and contains only a few pus cells. When these conditions are reached, the patient is likely to do better under general and sexual hygiene than under any local treatment. Indeed, one often sees such cases, in whom constant local treatment only aggravates symptoms, which would subside if left to themselves.

Vaccine Treatment.—Until the principles of vaccine therapy and the efficiency of the various bacterins and sera are more clearly understood, it seems quite hopeless to endeavor to bring order out of the chaos of conflicting competent observations concerning the use and value of this form of treatment.

I have employed vaccines, both stock and autogenous, but have failed to derive any great benefit from them, excepting in the treatment of epididymitis and gonorrheal rheumatism.

Inasmuch as the method of manufacture and strength of these preparations are never twice the same, it is impossible to give formulæ which can be generally applied. Each preparation should be employed according to the rules laid down by the laboratory from which it comes.

Treatment of Relapsing Prostatitis.—Certain patients who have suffered from severe prostatitis in the course of gonorrhea (and some whose original prostatitis was not due to the gonococcus) suffer from time to time from relapses which may be characterized chiefly by urethral discharge, or by outbreaks of chills, fever, and pyuria. Such attacks are usually brief and may be separated by months or even years. They are curable by prostatic massage, which should be given in courses of a month or two, with increasing intervals, until the patient has been watched and found free from tendency to relapses for at least a year.

Treatment of Retention Due to Chronic Prostatitis.—Exceptionally the result of chronic posterior urethral and prostatic catarrh is sclerosis either of the whole posterior urethra or of the neck of the bladder, which results in partial retention of urine by the same mechanism as that of prostatic hypertrophy. Such cases can only be cured by division of the neck of the bladder, which is preferably done through a perineal incision with the galvanocautery of Chetwood or Bottini.

URETHRAL STRICTURE

Prophylaxis.—To prevent traumatic stricture, perineal section should be done at the time of injury. To prevent gonorrheal stricture, every effort should be made to minimize the intensity of the inflammation, for, although stricture may result from chronic mild catarrh of the urethra, such stricture is usually readily dilated, while dense and unmanageable strictures are the result of intense urethritis or periurethritis.

Curative Treatment.—To cure a urethral stricture is not always possible. Stricture of the urethra anterior to the penoscrotal angle may be cured by dilatation to 32 or 34 F., or by cutting to this size. But the more common and more troublesome strictures of the deeper portions of the anterior urethra, notably those in the region of the bulb, while they may be controlled by dilatation, are often incurable, and will relapse after an interval of months or years in spite of any treatment.

Resection of the urethra (Cabot's operation) may achieve a permanent cure. It certainly changes intractable, resilient strictures into manageable ones. But many years must elapse before we can be sure that complete cure can be obtained even by this operation.

With this possible exception, stricture should always be treated by dilatation rather than by incision, and operation should be looked upon only as the means of opening a stricture which cannot be dilated or of curing some of the complications of stricture. After operation the sound is required as much as before.

The treatment of stricture may be considered under the following captions:

1. Stricture of large caliber.
2. Stricture of small caliber.
3. Stricture admitting only a filiform.

4. Stricture complicated by retention.
5. Impassable stricture.
6. Traumatic and resilient stricture.
7. Inodular or indurated stricture.
8. Stricture complicated by prostatitis (irritable stricture).
9. Stricture complicated by false passage.
10. Stricture complicated by periurethritis or prostatic abscess.
11. Stricture complicated by acute pyelonephritis.
12. Stricture complicated by fistula.

Treatment of Stricture of Large Caliber.—A stricture of large caliber is one which will admit a 20 F. sound. Such strictures, if not complicated, are to be treated by dilatation with steel sounds or dilators. If the stricture does not dilate it is to be treated as a resilient stricture (see below). If the passage of instruments excites inflammation or chill, the treatment is that of irritable stricture.

The passage of sounds into the urethra should always be followed by antiseptic washing with silver nitrate (1:10,000), or potassium permanganate (1:5,000), or by instillation of silver nitrate (0.2 to 0.5 per cent.) into the posterior urethra. I employ the latter treatment, as it seems not only antiseptic, but also helps to close up any minute abrasions that may be made in the urethra by its mild cauterizing effect. Furthermore, unless in an emergency, it is wiser to precede the passage of the sound by the administration of hexamethylenamin (gr. x, t. i. d.) for 2 days, until the temper of the urethra is known, after which the antiseptic may be dispensed with.

Finally, and above all, the sound must be passed gently.

The first operation upon a stricture should consist in the passage of a moderate-sized sound (20 F.). If this passes, it is wiser not to pass another instrument until the effect of this first instrumentation can be judged. If the sound does not pass, the stricture may be classed as one of small caliber. The patient is told to return in 3 to 5 days, and then a sound is passed, either of the same size or one size less than that passed on the previous occasion. If this passes readily, an instrument 2 sizes larger may be introduced, and, if this also passes without much force or bleeding, an instrument 1 or 2 sizes larger is introduced, and the operation closes with an instillation of silver nitrate. Thus the dilatation proceeds with intervals of at least three, and preferably 5 or 6, days between each passage of sound. Thus, with very gentle dilatation, we hope to gain from one to three numbers of the French (Charrière) scale on each occasion.

If the stricture responds kindly to dilatation, the first check is the size of the meatus. A meatus so small that there is a distinct pocket behind it, as indicated by a probe inserted into this, should be cut. But a normal meatus (which varies in size from 27 to 32 F.) is usually the standard for the limit of dilatation. When the stricture has been dilated to this size, the intervals are increased to 2, 4, 6, 8 weeks, and, if still there is no tendency to recontract, to 3 months, 6 months, and a year.

A stricture anterior to the penoscrotal angle that does not recontract after an interval of a year may be regarded as cured.

But deeper strictures require the passage of a sound once a year for the remainder of the patient's life, to insure against relapse.

This sounding can, of course, be performed more skillfully by the physician than by the patient, but, inasmuch as no patient was ever known to return year after year for the passage of a sound, it is only fair to recognize this human weakness and to instruct the patient how to pass a full-size sound upon himself. After having boiled it, and thoroughly washed his hands and penis, he celebrates with this instrument the advent of each Fourth of July or New Year's Day.

If, on any of these happy anniversaries, he is not able to introduce his instrument, he should apply at once to a physician for relief.

In some instances, however, the size of the meatus is not an adequate gage of dilatation. In such cases the stricture relapses within the year and dilatation must be carried to a higher point with a Kollman dilator, the limit in these cases being that size at which the stricture does not relapse after an interval of a year.

Treatment of Stricture of Small Caliber.—A stricture of small caliber is one that will admit a 10 F. instrument, but will not admit a 20 F. Such strictures are to be dilated with woven bougies according to the rules laid down above until these have reached the size of 20 F., when steel sounds continue and complete the cure.

Treatment of Stricture Admitting Only a Filiform.—Strictures that will not admit a 10 F. bougie may be classed under this heading. Such strictures are very frequently impassable, irritable, or resilient, and must be treated accordingly. But the proper employment of filiform instruments reduces the number of impassable strictures to a very small one.

The choice of instruments to dilate a filiform stricture is one of the most delicate tests of the experience of the urologist. All filiforms should be of the smallest possible caliber. A truly threadlike instrument often passes where another with a bulb the size of the head of a pin will not pass. Whalebone filiform bougies, such as are commonly employed in this country, should be selected with reference to their fineness and smoothness. The tips of these may be bent at an angle by immersing them in collodion and then bending the tip to the desired angle, and holding it in this position until the collodion dries. The outfit should include at least one-half dozen instruments.

As followers to the filiform, one should have two tunneled silver catheters, and at least two tunneled sounds, sizes 10 and 14 F. The eyes of such instruments should be large enough to slip readily over the filiforms. The Banks bougie, with its filiform tip enlarging to a thick shaft, makes a convenient substitute for the filiform and guided sound, since it can be introduced with much less danger of tearing the urethra.

Woven filiforms to screw on to a following catheter or bougie are made by various French firms; their disadvantage of being less stiff and less durable than the whalebone instruments is counterbalanced by the advantage of their fineness and smoothness. These instruments are also made with a copper wire core, which adds to their rigidity and gives the additional advantage that they can be bent at the tip to any desired angle.

Introduction of Filiforms.—Filiforms are apt to catch in the urethral folds and crypts, both in front of and behind the stricture. The following maneuvers are employed to overcome this difficulty:

1. When an instrument catches, partially withdraw and slightly rotate it, pushing it forward while making the rotatory movement. This device rarely fails in finally engaging the instrument in the orifice of the stricture, especially if the filiform point be bent or twisted in any direction (spiral or zigzag), so that its extremity may lie outside of the axis of the shaft of the instrument.

2. An excellent method of finding the orifice of a stricture consists in cramming the urethra full of filiform bougies, engaging their points in all the lacunæ and false passages, and then trying them, one after another, until that one is pushed forward which is presenting at the orifice of the stricture, when it will at once engage.

3. If the point of the filiform passes the stricture, but catches in the prostatic urethra, it may be lifted into the bladder by a finger introduced into the rectum.

4. If the stricture is a single band, the face of which may be reached by the urethroscope, this instrument is introduced, the stricture wiped with adrenalin until it ceases to bleed, and a filiform then introduced, guided by direct ocular observation. This maneuver rarely succeeds where other means fail.

If the filiform is introduced only after long and persevering effort, the question arises whether one may let the patient go after dilating the stricture to 10 or 12 F., or whether it is preferable to tie in a filiform for two or three days until the next instrumentation (in which case the patient should remain in bed), or whether immediate perineal section should be done. As a rule, the first course is safer. But complicated cases may require one or the other alternative, especially since a single success in passing a stricture by no means implies that one will ever get through it again.

After the successful introduction of the filiform and dilatation, this should be repeated every third and fourth day until the stricture is sufficiently dilated to take woven bougies in increasing sizes.

The Treatment of Retention.—Acute retention of urine from stricture may usually be relieved by the passage of a filiform, followed by a tunneled or guided catheter, as described in the preceding section. Two difficulties may arise in this connection: either the stricture may prove impassable or the retention, when once relieved, may recur. The treatment of impassable stricture is described in the succeeding section. Recurring retention is exceedingly annoying, and may continue until the stricture has been dilated as high as 20 F. In such cases, even though the recurrence of retention is obviously due to a marked congestive tendency, it is my practice to push dilatation very rapidly up to 20 or 22 F., reaching this in two or three sessions at three or four-day intervals. After this dilatation is attained, one may go more slowly, since the possibility of retention has been overcome.

The Treatment of Impassable Stricture Without Retention.—The treatment of this condition exercises the judgment of the surgeon as to how long he may coax the stricture in the hope of introducing a filiform through it without undue risk to the patient. The various renal function tests, notably the phenolsulphonephthalein test, are of great value in this connection. If the patient is afebrile, and the renal function is good, one makes many efforts before giving up and having recourse to perineal urethrotomy. But if the patient has chills or if the kidney function is bad, urethrotomy should be performed at once, and although external urethrotomy without a guide is a difficult and dangerous operation in unskilled hands it is to be remembered that the skillful surgeon can offer much more brilliant hopes of recovery with this than by indefinitely prolonged efforts to get through an impassable stricture.

Treatment of Impassable Stricture With Retention.—In this condition the emergency is acute. The patient must be relieved immediately. If filiforms fail to pass, the patient may be put in a hot sitz bath at a temperature of about 100° F., and the hot water kept running so that the temperature is gradually increased as much as the patient can bear. This bath is continued for about ten minutes. If it causes faintness and nausea, it is all the more likely to cause relaxation. The patient is urged to try to urinate in the bath. Immediately after the bath he is put to bed with hot blankets about him, and again urged to urinate. If this treatment fails the bladder should be aspirated above the pubes. This may be repeated several times. Yet suprapubic aspiration is only an emergency method, and if within 12 or 24 hours it is not followed by sufficient relief of congestion on the surface of the stricture to admit the passage of the instruments, the patient should be cut in the perineum.

Treatment of Traumatic and Other Resilient Strictures.—Traumatic strictures are almost invariably resilient, i. e., they either refuse to dilate or, once dilated, promptly recontract. Resilient stricture (whether traumatic or not), if anterior to the penoscrotal angle, should be cut to 32 or 34 F. with the Otis or Maisonneuve urethrotome. If posterior to this point, such strictures should be resected, for, if they are simply cut, they are likely to reconstruct quite as badly as ever. The Cabot operation for resecting is the one I prefer.

Treatment of Inodular or Indurated Stricture.—Strictures complicated by lesions of scar tissue in and about the urethra require resecting after the scar tissue has been cut away.

Treatment of Irritable Stricture.—By irritable stricture is meant that type of stricture the treatment of which by sounds is followed by chills, acute urethritis, or bleeding. The bleeding may arise from the stricture, but the local or inflammatory reaction always, and the bleeding usually, are due to inflammation behind the stricture, generally in the form of prostatitis. Under such circumstances, if the stricture is not so tight as to prohibit delay, it is better to treat the patient first by hexamethylenamin and rectal injections, followed by gentle but persistent prostatic massage; next by instillations of silver nitrate, then to resume again the gentle passage of sounds. If, in spite of all this, the reaction reappears, perineal section should be done at once.

Treatment of Stricture Complicated by False Passage.—If a false passage has been made in the effort to get by a stricture, and the patient's condition permits, no further instrument should be passed for two weeks. At the end of that time sounding may be gently resumed, with the hope that the false passage has healed. But, if the stricture requires immediate dilatation, false passage may make the stricture an impassable one, to be treated as above described, while, if there are chills or other complications, these require prompt perineal section. In avoiding an old false passage it is necessary to locate its orifice first by noting in which direction the sound is deflected as it enters this. Having noted this, attempts at dilatation are subsequently made with the point of the instrument deflected away from the orifice of the false passage. False passages on the roof of the canal are so uncommon that it is almost a universal rule that they may be avoided by hugging the roof of the urethra with the point of the instrument.

Stricture Complicated by Periurethritis.—ABSCESS OF INFILTRATION.—The presence of any periurethral abscess, deeper than the penoscrotal angle, or of prostatic abscess, or urinary infiltration, calls for perineal section with evacuation of all pockets, free incision, and drainage and division of the stricture.

Treatment of Stricture Complicated by Acute Pyelonephritis.—The kidney must be drained either by a retained catheter or by perineal section. The tube should remain in place until the temperature touches normal. This failing, nephrotomy is required.

Treatment of Stricture Complicated by Fistula.—The chief aim in the treatment of fistula is to remove the impediment to urination—in most cases to dilate the stricture. This done, the fistula will usually close itself; but, as long as the urethra is obstructed, the urine will seek the freest outlet, viz., the fistula.

Indurated fistula is usually associated with resilient or impassable stricture. To cure it, all the scar tissue about the fistula and the urethra must be excised and external urethrotomy performed.

POSTGONORRHEAL NEUROSES

Two types of neuroses may result from gonorrhea. These are: sexual neuroses and painful neuroses.

Precisely the same types of neuroses are seen in persons who never had gonorrhea, and, whether following gonorrhea or not, their pathological basis consists in inflammation of the whole posterior urethra, or of the prostatic utricle alone, hypertrophy of the verumontanum, chronic prostatitis, or chronic seminal vesiculitis. Furthermore, there is in each case a psychic element of greater or less importance, and, finally, the pain may be falsely attributed to lesions in or about the posterior urethra, when it is actually due to neurosis or to one of the forms of intestinal indigestion commonly associated with indicanuria.

Therefore, before beginning treatment, all these elements of the case must be carefully studied, especial attention being paid to the neurotic element, for, in many instances, it will be found that fear of grave disease is the chief element in the case and the patient only requires to be reassured in order to be willing to put up with the mild inconvenience of which he complains. In other instances, treatment of the digestive disturbance will relieve the pain; in others, general hygiene will succeed, and, finally, a large proportion of the patients who complain of feeble erection and premature ejaculation require for their cure rather a study of their peculiar sexual aberration than any local treatment.

There still remain many cases, however, whom this treatment fails to relieve. For most of these cases more can be done by treatment of the verumontanum through the posterior urethroscope than by any other form of local treatment. The straight tube may be used (I prefer the Swinburne posterior urethroscope). Through this applications of 20 per cent. silver nitrate or 50 per cent. liquor hydrargyri nitratis are made once in ten days. The application may be made on a very fine cotton swab directly to the verumontanum, or on a large swab and followed by salt solutions to prevent excessive irritations. For similar cases Dr. Geraghty has obtained good results by injecting a few drops of 1 per cent. silver nitrate into the utricle by means of a special urethroscopic syringe.

Oversensitive patients may be made distinctly worse by these methods of treatment, and for them the gentle passage of sounds and the employment of instillations of nitrate of silver are more satisfactory. Exceptionally, prostatic massage by the finger, or by a vibratory massage instrument introduced into the rectum, or by the galvanic or high-frequency current are of use. But, as a rule, the effect of such treatment is suggestive rather than physical, and, first, last, and all the time, sexual and general hygiene must be borne in mind.

CHAPTER II

SPERMATORRHEA AND URETHRORRHEA

E. L. KEYES, JR.

At the termination of a chronic urethritis the patient may be left with a slight urethral discharge, mucoid and sticky in character, either absolutely clear or slightly opalescent, and sometimes so slight as to cause only an adhesion of the lips of the meatus to each other when the patient awakes in the morning; sometimes sufficient to cause a urethral discharge throughout the day. Examination of this discharge reveals a body of mucus entangling bacteria, urethral epithelium, and an occasional leukocyte; but no gonococci; no pus. Such a condition is commonly known as urethrorrhea or prostatorrhea. The distinction made between the two can be based only on the result of treatment. If the discharge can be cured by massage of the prostate, it may be put down as a prostatorrhea. If not, it should be classed as urethrorrhea, and is to be treated by the injection into the anterior urethra of astringents (pages 155 and 156) (with the proviso that such injections may do the patient harm instead of good), by the passage of sounds and dilators to as high a number as the urethra can bear, with the object of massaging the urethral glands, and by attention to the patient's sexual and general hygiene. In certain instances such discharges will only cease when the patient's hygienic condition is materially improved, all local treatment doing more harm than good.

Spermatorrhea is the title given to two distinct conditions: On the one hand, the patient's urine, when voided and examined under the microscope, may prove to contain spermatozoa. It is most unwise to call the patient's attention to this condition, as it is quite harmless, although the knowledge may fill him with strange fears.

On the other hand, the patient may extrude from his prostate and seminal vesicles a drop or more of semen when he attempts to have a movement of the bowels. Constipation, of course, increases the amount of discharge, which may be the source of considerable alarm, and which sometimes occurs under other conditions; thus it may follow the act of urination or any muscular strain. This condition is entirely harmless, as such. If the seminal fluid extruded is not mixed with pus it simply means that the muscles of the internal genitals are somewhat relaxed, and, while the amount of discharge may be materially diminished by regulating the patient's sexual affairs and constipation, and by occasionally massaging the prostate, it is unwise to depend upon physical measures for a cure, since, in spite of them, a slight discharge is likely to persist. This is harmless, and the patient must be instructed to disregard it.

CHAPTER III

IMPOTENCE

E. L. Keyes, Jr.

TREATMENT OF ORGANIC IMPOTENCE

Impotence may result from many congenital and acquired defects, such as hypospadias, tight urethral stricture, tabes, etc. Such impotence is, of course, only as curable as is its cause.

TREATMENT OF FUNCTIONAL IMPOTENCE

The treatment is threefold:

The Patient's Sexual Coefficient Must Be Discovered.—By the sexual coefficient is meant the amount of sexual power with which the patient is endowed by Nature. Mankind at large is possessed by the notion that, although men's noses and digestions need not all be cut of the same pattern, it is to be expected that the sexual capacity of every one should be all-embracing. Thus, while it is no disgrace to be dyspeptic about the stomach, it is to the last degree shameful to be dyspeptic about the genitals. Theoretically, such a distinction is absurd; but practically no man is willing to brand himself a sexual laggard.

In some way, by dint of enumerating emissions, copulations, masturbations, the physician must learn what ideal he can set before the patient. If a man has a natural sexual capacity for copulation only once a month, it is hopeless to try and tune him up to three times a night.

The Patient Must Be Encouraged.—The first point of encouragement must be to depress him by bidding him look for a protracted and relapsing convalescence. Then he must be made to understand that his sexual possibilities are just so great and no greater; and that, however well he may get, overstepping his allotted bounds will call down swift retribution upon him. Finally, he must really be encouraged to feel that his malady is a functional disorder, a dyspepsia, which, like other dyspepsias, is curable, but only at the cost of a prolonged fast. He must abstain from coitus, from masturbation, from lewd companions, from obscene thoughts and things. The more thoroughly he abstains the more certain his cure. Usually he will try to adopt halfway measures, caring more for his "pot of ale" than for body and soul together. But such a course may not be countenanced. The ideal of absolute purity must be forever set before him, and, as it were, hammered into him.

He Must Be Assisted Physically.—When possible, an entire change of scene presents the best opportunity for a man to get out of his old rut. Nothing could be better than a hunting or fishing trip. But, if this is impossible, tonics may be given him, and sounds, nitrate-of-silver applications, rectal douches, according to the choice of the physician. To some hyperesthetic individuals massage of the vesicles is so nearly a suggestion of the sexual act that I fear it is quite as likely to do harm as good. If a strong moral influence, as that of father, brother, or priest, can be brought to bear, so much the better.

But all these measures are frankly palliative. When a man has once got into the habit of concentrating his whole mind upon his sexual organs it is not to be expected that he should be entirely diverted to higher things. Chastity all can aim at, but celibacy is beyond the reach, beyond even the understanding, of the many. Hence, the proper cure for such a man, if he can be got into such a condition that he has an erection ever so rarely, is to instruct him in sexual physiology and hygiene, to acquire his confidence by sympathy, and to get him married, with the advice to attempt no intercourse, to be entirely frank and honest with his wife (who will more than equal him in timidity and ignorance), and, awaiting some morning when awaking with a vigorous erection, to accomplish coitus promptly, without delay, as a matter of imperious duty. The act once accomplished, the spell is broken. He knows he is a man, and his confidence in himself returns.

IV DISEASES OF THE NERVOUS SYSTEM

CHAPTER I

DISEASES OF THE SPINAL CORD

JOSEPH COLLINS AND EDWIN G. ZABRISKIE

TABES

Tabes, tabes dorsalis, locomotor ataxia, or posterior spinal sclerosis is a disease which depends anatomically upon a degeneration of certain sensory neurons, particularly those whose neuraxons form the posterior columns of the spinal cord and those which constitute the optic nerve. The disease is usually described as a sclerosis of the columns of Goll and Burdach; but the sclerosis is entirely secondary and is to be interpreted as the result of an effort of Nature to fill the vacancy left by the degenerated neuraxons. It may be defined as a degeneration of that area of the spinal cord constituted by the sensory neurons. Clinically the disease is characterized by a more or less progressive course, by an association of sensory, motor and trophic symptoms, which, taken together, are absolutely characteristic, and by its evolution in a more or less typical way.

Causes and Lesions.—Very little is known of the causation of tabes, save that it occurs predominantly in middle adult life in those who have had syphilis. Although opinion is not unanimous in regard to the syphilitic origin of tabes, it is almost universally conceded that from seventy to ninety per cent. of all tabes patients either give a history of syphilitic infection or bear unequivocal marks of its existence. The finished or terminal lesion of tabes is not, however, characteristically syphilitic; in other words, the decay of the posterior columns is not secondary to gummatous infiltration or any of the established types of luetic changes. The lesion is spoken of as parasyphilitic, to convey the idea that it is the result of the activity of syphilis, or of some noxious agency engendered thereby after the syphilitic poison has been deprived of the power to manifest itself in inflammatory reaction; but this conception is gradually losing ground, especially since serological investigations are furnishing evidence of the nature of the earliest changes. Oppenheim thinks it is conceivable that chemical bodies caused by the dissemination of syphilitic poison throughout the organism appear and produce a simple degeneration of special parts of the nervous system.

The recent investigations of Sicard, Widal, Plaut, Nonne, Hauptman, and Kaplan, on the Wassermann reaction of the serum, and the Wassermann reaction, globulin estimation, Fehling reduction, and cell count in the cerebrospinal fluid in tabes, have confirmed to a great degree the theory of a syphilitic origin of the disease, and have increased the percentage of cases with demonstrable syphilis very considerably. The most recent work on the subject is by Nonne. He insists that, although all cases do not give a positive Wassermann reaction in the blood, 60-70 per cent., and 5-10 per cent. in the spinal fluid if the original method (0.2 c. c.) is used, it is nevertheless positive in 100 per cent. if larger quantities of the fluid are used.

The disease occurs more frequently in males than in females; but it is not so infrequent among the latter as was formerly thought to be the case. Among people of the better classes the number of men is far greater than women. Sarbo found among the working classes of Buda-Pesth a proportion of 2 to 1. Mendel found 3 to 1, while Marie in a recent communication to the Paris Neurological Society believes that just as many women as men acquire tabes, but such is not our experience by any means.

It is met much oftener in private than in dispensary practice, and in the intellectual and upper classes than in primitive peoples and the lower classes, and occurs oftener in the Caucasian race than in the Ethiopian and Mongolian. Exposure to cold, frequent and prolonged fatigue, sexual excesses, intemperate use of alcohol and tobacco, poisoning by ergot and lead, the infectious diseases, and trauma have been considered etiological factors ever since the disease was first described by Duchenne in 1861. They are contributory factors of some weight, but only lead or ergot can rightly be said to cause symptoms resembling the disease. The French school headed by Charcot have unswervingly contended for the importance of a neurotic predisposition and the arthritic diathesis as factors in the occurrence of the disease. Our cytological investigations are proving conclusively that tabes is a syphilitic disease, and that it arises from syphilis as a brook flows from a spring.

Oppenheim, Erb, Nonne, Kalischer, Fournier, Babinski, and others report cases of tabes in individuals who denied having acquired syphilis, but whose parents had either had syphilis or tabes (conjugal type).

Heredity is of little importance. There is no doubt, however, that a defective nervous system, i. e., one incapable of resistance to disease processes, may be transmitted to one or more offspring. The neuropathic diathesis is a predisposing cause. Unquestionably fatigue and leg weariness, as from long standing, forced marches, and occupations requiring exhausting use of the legs, have something to do with precipitating or possibly, even, initiating the tabes. Thus the disease is seen oftener in persons whose occupations require them to be on their feet a great deal than in persons of more sedentary occupations. We have seen two cases of tabes develop suddenly in men who had had syphilis, upon their change from a sedentary occupation for camp life preparatory to the late war. Although traumatism may accelerate the progress of the symptoms, it has never been proved to be the cause of the disease, as has been urged by some. Anything that exhausts the peripheral sensory neurons and maintains the exhaustion is a predisposing cause of tabes.

The relation of fatigue, trauma, and sexual excess to the precipitation of tabes has become quite plain since the publication of Edinger's interesting hypothesis. He believes that people under the influence of certain poisons (in the widest sense), of which the syphilitic poison is the most common, may exhaust the nerve tracts that are in constant use. He bases this upon the Roux-Weigert theory of balance of individual parts in the organism, and assumes that all cells become weaker during activity because of the molecular loss. This loss is replaced during rest in normal individuals. In pathologic states, either because of faulty restitution or unusual functioning, a disturbance of the relation between function and restitution occurs, and degeneration ensues.

Although tabes occurs predominantly in middle life, there is practically no period at which it may not appear. Heredotabes has been reported in children from eight years upward; E. Mendel reported recently the case of a man infected at 70 years, who four years later developed tabes. As a rule, it appears within 5 to 20 years after infection, but it may appear within one-half year, or 50 years after the chancre.

The morbid changes found in tabes are widespread and occur in central and peripheral parts of the entire nervous system; but the most constant lesions are: degeneration of the posterior columns, Lissauer's marginal zones, posterior horns, Clark's columns, posterior roots, spinal ganglia, and thickening of the piaarachnoid covering the posterior surface of the cord and posterior roots. If the disease has been of long standing a secondary sclerosis of the glia occurs, but this is always secondary, and it depends, as we have said, chiefly upon the length of time the disease has existed.

The posterior columns are usually affected only in part and in proportion to the number of roots affected. As the disease selects the lumbosacral and lower dorsal regions as the point of greatest predilection, we naturally find at these levels a fairly widespread degeneration in the fibers of the posterior columns. As we ascend to higher levels healthy fibers crowding the degenerated ones toward the median line are found, so that in a mild, well-limited lumbar lesion the degenerated fibers in the cervical cord will occupy the columns of Goll only. Frequently we find degeneration occurring at different levels, so that we may find degeneration in the lumbar cord, with isolated degeneration in the cervical levels, in which the descending tracts of the posterior columns are conspicuously affected.

The changes in the posterior root ganglia have until recently occasioned considerable discussion, and as long as the Nissl method was depended upon there was no unanimity of opinion concerning the changes in these structures. Marinesco, Nageotte, and Köster found mild chromatolysis, fatty degeneration, pigmentation, increase of the endothelial capsule, and shrinkage of the cell. With the advent of the more delicate neurofibril stains of Cajal and Bielchowsky more definite and constant changes in the posterior roots and ganglia have been described. These consist in granular degeneration of the intracellular fibrils, vacuolization, variations in the size and number of fibrils of the axones, and a peculiar nodular arborization of the axone which Nageotte believes to be regenerated fibers. Certain it is that, if the ganglia are obtained in the early stages of the disease, they present definite changes similar to those produced experimentally.

The other structures of the central nervous system usually affected are the descending root of the trigeminus, the Gasserian ganglion, fasciculus solitarius, optic nerves, the ciliary ganglion, peripheral nerves, sympathetic, occasionally the fibers and nucleus of the oculomotor or abducens, and sometimes the hypoglossus nucleus. In cases where the disease has been complicated by muscular atrophy a corresponding lesion in the anterior horn cells has been found. In a few instances well-marked degeneration in the lateral pyramidal tracts has been found without clinical evidence to betray it.

The changes in the blood vessels are not at all constant, and, whether they are in the meninges or in the cord itself, the lesions are usually not characteristic of syphilis, but appear to be local reactions dependent on the chronic process in the midst of which they are. In early, fresh, uncomplicated cases there are usually no vascular changes worthy of note. The tangential fibers of the cortex nearly always disappear, and low-grade vascular infiltrations are usually present.

The pathogenesis of tabes is still very obscure, and, in spite of all the work hitherto accomplished, there is no unanimity of opinion concerning the primary lesion. The first important step in the right direction was taken by Nageotte, 1894, who described a low-grade meningitis at the junction of the anterior and posterior roots on the proximal side of the ganglion. This view was not generally accepted at the time, as the majority of workers were unable to confirm his findings. Prior to 1894 various theories appeared from time to time, some advocating a primary sclerosis, others making the lesion dependent upon vascular changes in the roots and posterior columns, and with the advent of the Nissl stain a primary degeneration of the spinal ganglia was held by others to be the starting point of the pathological changes. Nageotte described, in addition to the meningeal process, an actual neuritis, infiltration of the perineurium, perivascular infiltration, and the presence of infiltrating lymphocytes and plasma cells in the posterior roots. The chief objection to this view lay in the inconstancy of these changes, and also in the fact that, while the anterior roots are just as much invaded by this process, the fibers do not show similar degeneration. In 1903 Marie and Guillain brought forward a hypothesis, based on experimental and histologic grounds, that tabes is really a lymphangitis of a system which includes the posterior columns, posterior horns, posterior roots, and the overlying meninges. As their reasons, especially the experimental proofs, were not convincing, the theory has received little attention, although so competent an observer as Oppenheim is inclined to look on it with favor. The most recent work seems to point to a primary degeneration of the posterior root fibers themselves, with possibly the implication of the ganglion cells as well. This is supported both by studies with the Bielchowsky method of staining the neurofibrils, and also by some recent experiments on the effects of inoculation of dogs with trypanosoma brucei by Spielmeyer. He was led to these experiments by the fact that the trypanosomes of the sleeping sickness really belong to the spirochetes and are closely related to the spirochæta pallida. After 9-10 weeks he found an elective process in the posterior roots, which was beginning to appear in the posterior columns, sensory trigeminus tract, and optic nerves.

Course of the Disease.—Tabes dorsalis is usually a progressive disease up to the point of complete destruction of the posterior columns of the spinal cord, which coincides clinically with the complete uselessness of the patient for any purpose save as a manifestation of vitality. The course of the disease may be very slow, but, if untreated, each succeeding year usually finds the patient a little more incapacitated. Despite this it should not be forgotten that, clinically, tabes is a recoverable disease. Unfortunately complete recovery does not often occur.

The clinical course of the disease is divided into: 1. A preataxic period, with its accompaniment of paresthesias, lancinating pains, disturbances in the urogenital sphere, and neurasthenic symptoms. 2. Ataxic period, characterized by motor incoördination, loss of tendon jerks, objective sensory disturbance, hypotonia, and immobility of the pupil on exposure to light; and 3. A terminal stage attended by general muscular weakness and impairment of vegetative functions, in addition to the symptoms before mentioned, with the exception of pain, which, as a rule, completely leaves the patient in this stage. The duration of the disease is from ten years to half a lifetime. Certain cases terminate fatally within a few years, but it is very questionable whether these can be regarded as true tabes.

Diagnosis.—Ordinarily the diagnosis of tabes is not difficult; even in the earlier stages the presence of some of the signs: absent knee jerks, Argyll-Robertson pupils, lancinating pains, etc., point positively to the nature of the disease. Formerly it was often very difficult to distinguish between tabes and polyneuritis, in which ataxia, absent reflexes, and lancinating pains were present, and the differentiation from diabetes complicated by neuritic manifestations was also very uncertain. These difficulties, however, have to a great extent been removed by the studies of the cerebrospinal fluid, and also by the Wassermann reaction. The investigations of the cerebrospinal fluid by Sicard, Widal, Plaut, Nonne, Kaplan, and others have established fairly definite types, which give us a much clearer insight into the manner in which the cord is being attacked. In attempting the diagnosis the spinal fluid should be subjected to four tests, i. e., Wassermann reaction, cell count per c. mm., globulin content, and Fehling reduction. These four tests, together with the Wassermann reaction in the blood, vary in different types of organic disease of the nervous system in such a way as to be a valuable adjunct to the clinical picture. The most constant variation from the normal cerebrospinal fluid is in the number of cells per c. mm. Normally these should not be above 8 to 10, and are, for the most part, lymphocytes. In all parasyphilitic diseases of the cerebrospinal axis they are increased more or less, according to the type of the disease. The Wassermann reaction is positive in a fair proportion, and absent in others. The globulin content is usually increased, and Fehling's solution is reduced.

The Wassermann reaction in the blood is positive in some and frequently absent in others. Nonne, in the September, 1911, issue of the *Deutsch. Zeitschrift. für Nervenheilkunde,* reviews the present status of our knowledge of these reactions, and comes to the following conclusions:

Typical findings in

I. Paresis or Taboparesis.—Wassermann reaction of blood serum positive in almost 100 per cent. Lumbar pressure increased. Globulin increased, 95-100 per cent. Lymphocytosis, 95 per cent. W. R. in cerebrospinal fluid positive in 85-90 per cent., original method (2 c. c. of fluid). W. R. in cerebrospinal fluid positive in about 100 per cent. if larger quantities of the fluid are used.

II. Tabes.—W. R. in blood positive in about 60-70 per cent. Lumbar pressure increased. Globulin content increased in 90-95 per cent. Lymphocytosis in 90 per cent. W. R. in fluid positive in 5-10 per cent., original method (2 c. c. of liquor). W. R. in fluid positive in about 100 per cent. if larger quantities of the fluid are used.

III. Cerebrospinal Lues.—W. R. in blood positive, 80 per cent. Lumbar pressure increased. Globulin content almost always increased. Lymphocytosis always positive. W. R. in fluid positive in about 10 per cent., original method. W. R. in fluid positive if larger quantities are employed.

From the foregoing it will readily be seen that we have positive laboratory data which is a very valuable aid in diagnosticating tabes. In the case of polyneuritis or diabetes, we find the Wassermann reaction negative in blood unless syphilis be present adventitiously, and the fluid should be negative in all tests. The real difficulty, then, lies in a proper differentiation of paresis, tabes, and cerebrospinal lues. Tabes may be complicated by neurasthenic or emotional symptoms, which make one suspicious of paresis or taboparesis in the early stages. The reactions will usually help us in this instance, since in tabes the Wassermann reaction of the cerebrospinal fluid is frequently negative unless larger quantities are used, and unless it be of the hyperlymphocytic type; the cells average 8-60. In paresis they average 8-90. The Wassermann reaction in the blood is almost invariably positive in true paresis, while it is often negative in tabes. Antiluetic treatment affects these reactions, as one would naturally suppose, and this probably explains the large number of negative Wassermann reactions found in tabes. The reaction does not seem to be affected, however, by any treatment whatever in general paresis, and it persists, as a rule, to the end. The lymphocyte count and globulin are most affected by treatment, the cells diminishing in number, and the globulin excess becoming normal.

The early diagnosis of tabes is most important, since a prompt recognition often means arrest of the morbid process, and hence the prevention of much misery for the patient. The only safeguard against errors of diagnosis is most meticulous physical examination of every patient who has any of the recognized symptoms of the disease. The existence of one cardinal physical sign plus typical changes in the cerebrospinal fluid is, in our judgment, sufficient data for a diagnosis. The Neurological Society of Paris recently discussed the occurrence of monosymptomatic tabes. The general sentiment was that, as there was no one pathognomonic sign, there could be no such type. Clinically this may be true, but the study of the cerebrospinal fluid has shown us that it is possible to have one symptom, either pains or Argyll-Robertson pupils, together with spinal fluid reaction indicative of tabes.

Occasionally cervical tabes presents difficulties of diagnosis. We may find knee and ankle jerks intact, no Romberg nor bladder disturbances, but the Argyll-Robertson pupil is invariably present; lancinating pains in the arms and neck, anesthesias of the upper trunk, and absence of the reflexes of the upper extremities are the diagnostic points to be borne in mind. Déjerine has described conus tabes, in which for many years the only signs are anesthesia of the anus and perineum, loss of sphincter control, and sexual impotence.

Symptoms.—The symptoms of tabes vary greatly, and one might say that the clinical appearances of the disease are almost protean. In every case, however, there is always a group of symptoms and signs which, taken together, are quite distinctive. Two or three of them are invariably present in every case, and, as they constitute the earliest signs of the disease, they will be considered first. They consist of: 1. Absence of tendon reflexes, i. e., knee jerks and ankle jerks. 2. Argyll-Robertson pupil. 3. Romberg's sign. 4. Lancinating pains. 5. Diminution or loss of cutaneous sensibility. These are the most constant of the earlier signs, but we frequently find a history of transitory diplopia or sudden loss of sexual power preceding the other symptoms by many years.

1. Absence of tendon reflexes, i. e., knee jerks (Westphal's sign) and Achilles jerk, is the most constant sign of tabes, and is often present a long time before other signs appear. It varies greatly in the early stages, and may consist of inequality of the jerk, absence of one knee jerk with diminution of the other, absence of both ankle jerks with diminution of one knee jerk and no change in the other, or there may be alternation, that is, one knee jerk absent with retention of ankle jerk on the same side, while the opposite side shows absent ankle and present knee jerk. At first the knee jerks may be diminished to such a degree that they can be elicited by reinforcement only. This is known as Jendrássik's phenomenon, and consists in removing the patient's attention to something else by having him pull his hands forcibly apart, counting aloud, or by having him recount his story. In testing the knee jerks it is important to have the quadriceps tendon relaxed and slightly stretched, either by having the patient sit with the knees crossed, the foot swinging free, or else by having him lie down while the examiner lifts the knee gently until it is partially flexed. A sharp blow is then struck over the patellar bursa with some blunt instrument. The Achilles jerks are best obtained by having the patient kneel on a soft cushion placed on a chair so that the weight rests on the knees alone, the feet hanging over the edge of the chair, or else the patient lies prone on the bed or couch, the legs are flexed, and the Achilles tendon is slightly stretched by passively flexing the foot. Care must be taken in eliciting the ankle jerks not to strike the tendon at its insertion in the calf muscles, because if the muscle is struck a response will be obtained although the true tendon reflex may be absent.

2. The Argyll-Robertson pupil is almost as constant as Westphal's sign. It consists of loss of response to direct illumination with preservation of the pupillary contraction on convergence of the eyes gazing at a distant object. A complete loss of contraction to light is not invariably present, nor is it necessary for the determination of the phenomenon. Sometimes there is merely a sluggish contraction of the pupil, or it may respond only to strongly concentrated beams of light. The best method is to bring the patient close to a moderately well lighted window and then shade the pupils with the hand. When the hand is quickly removed the pupil will contract promptly if the normal reaction is present. If the

patient is in bed the test may be made with a lighted match, a candle, or a small pocket electric lamp. If the latter is used care must be taken not to bring the light nearer than 6-12 inches to the patient's eye. Sluggish or absent reaction has been noted in chronic alcoholism, chronic lead poisoning, and meningitis, but we desire to make emphatic statement of the fact that we believe it to be a manifestation of disease solely the outcome of syphilis. The shape of the pupils is likewise an important sign in tabes. In the majority of cases they are oval, pyriform, or otherwise non-circular.

3. Romberg's sign consists of inability of the patient to stand securely with the feet close together when the eyes are closed. There is considerable discussion concerning the cause of this phenomenon, and the former view that it is due to blockings of impulses conveying the sense of attitude, i. e., position sense, deep muscular sense, joint sense, etc., has been recently combated by Bonnier and others, who attribute the phenomenon to disease of the vestibular apparatus, or the labyrinth itself. That this is not always the case has been shown by Frenkel, Jacquod, and Förster in cases of severe anesthesia of the soles. We may also find it in certain forms of peripheral neuritis of the lower extremities, such as chronic arsenical neuritis or alcoholism, where there are no evidences of vestibular disease. In some instances the swaying is so slight that we may be in doubt as to whether a true Romberg exists. We may then have the patient crouch or slowly sink to his feet, or have him stand on either foot alone. The uncertainty of station is always so conspicuous when a true Romberg exists that there can be no mistaking it.

4. Lancinating pains, lightning pains, electric or spot pains, are extremely characteristic. They are frequently called rheumatic pains by the patients, and often treated as such by unobserving practitioners. They usually occur in paroxysms, are described as sharp jabs, knife-like in character, and rarely spread over large areas. They may be localized in one particular spot (spot pains), and occur at rhythmic intervals that almost completely demoralize the sufferer. They are frequently attended by cramp-like contractions of the muscles and followed by great tenderness of the parts. The arms and legs are most often affected, and in some instances the intense pains will be limited to one single spot on any part of the limb, although the feet most usually are the seat of this particular form. Sometimes the girdle sensations may be accompanied by girdle pain which, when it occurs, is usually very severe. The groins also are favorite places for the appearance of these pains, whereas the trigeminus and upper cervical nerves are only occasionally affected. This, in view of the frequency with which the sensory root of the trigeminus is affected, seems strange. Very rarely the paroxysms are accompanied by temperature increase, vasomotor crises, local edema, agglutinations.

5. Anesthesia, analgesia, delayed sensation, and paresthesia are

constantly seen early in the disease. Frequently analgesia or hypalgesia of the lower extremities with preservation of all other forms of cutaneous sensibility is seen, and in many instances it extends upward to the upper dorsal levels. The French school (Déjerine, Babinski, etc.) consider this phenomenon of great diagnostic importance, however slight it may be. Déjerine considers it of diagnostic significance whenever the inner sides of the thighs and arms are less sensitive than the outer, since this is a reversal of the normal state. There are certain areas or zones in which these disturbances usually appear: the lower extremities, brachiomammary, urogenital, and cephalic zones. The sensory disturbances, on the other hand, may consist simply in delayed transmission of the impulse, so that 10-15 or 30 seconds after the examining finger or instrument has been removed the patient perceives the touch. It is most frequently found in the lower limbs, and only very rarely in the arms or trunk. Paresthesias are very common and usually take the form of tingling, numbness, or burning sensations. They are found most frequently in three areas: the girdle sensation about the abdomen, the outer side of the calf and foot, and the ulnar distribution. Paresthesias of the trigeminus, tinnitus, tickling sensation in the larynx, have also been described. Paresthesia, or loss of vibration sense in the long bones, pelvis, and cranium is often encountered, although this usually appears later. Loss of deep muscular sensibility also appears later, and only after the progress of the disease has become quite marked. It is characterized by the inability of the patient to recognize with closed eyes movements of joints executed passively, or to describe the attitude in which the limb has been passively placed. This can also be tested by having the patient simulate with one limb the attitude in which the other has been put, or by having him point at the great toe with the forefinger of the opposite hand and repeat this with the leg in various attitudes. It is absolutely essential for this test that the muscles of the limb examined be absolutely relaxed.

As the disease progresses, the ataxia begins to appear, and the patient realizes that there is difficulty of locomotion. This phenomenon usually appears insidiously at first, as a slight stumbling when walking over uneven surfaces or after stepping off or on curbs, a sudden giving way at the knees when walking down steps, and soon he realizes that it is necessary to watch the ground carefully while walking. The ataxia may never progress beyond this point, but in well-developed cases the gait becomes unsteady, staggering, slow, the heel planted down first or the whole foot slapped down in an awkward, ungraceful manner. In advanced stages it becomes impossible to walk without support. The more severe forms of ataxia are always accompanied by a marked degree of hypotonus of the muscles and tendons. This allows hyperextension or hyperflexion of the joints, contributes to the exaggerated joint excursions of the ataxic gait, and if not corrected tends to produce marked deformities of the knee and ankle joints. It may become so profound as to permit the most grotesque contortions, such as flexing the extended leg on the trunk until the feet meet behind the head, or extension of the leg until it forms an angle of 60 degrees with the thigh.

Ataxia of the arm is rarely so well marked as in the legs, but betrays itself in the inability of the patient to perform delicate, coördinated acts, i. e., writing, buttoning the clothes, drawing, etc. It can best be elicited by having the patient attempt to touch or grasp objects when the eyes are closed. When these movements are attempted they are performed awkwardly, and the finger, instead of attaining its goal with sureness, sways, runs by the object, and usually only reaches it by feeling about. Ataxia of the eye muscles is seen in the transitory diplopias that occur during the course of the disease.

Impairment of bladder function is almost constantly seen in well-developed cases. It may consist in difficulty in starting the stream so that pressure through the abdomen and diaphragm becomes necessary. There may be inability completely to empty the bladder through lowered tone of the vesical wall, or there may be sphincter hypotonus causing inability to hold normal amounts of urine in the bladder. On the other hand, there may be complete or partial incontinence as a result of anesthesia of the sphincter. The rectum may be similarly affected, but obstinate constipation is most frequent.

Of the cranial nerves the optic is most frequently affected in the form of bilateral simple atrophy, slowly progressive, which leads in many instances to complete amaurosis. The visual fields are usually concentrically contracted, but occasionally central scotomata are observed. Von Graefe reported bitemporal hemianopsia in a case of tabes, and we have recently had a similar case under observation. The ocular muscles are frequently affected, but it is rarely permanent. The paralysis may affect the abducens or some of the branches of the oculomotor. A low degree of ptosis, which can be overcome by great effort, is often seen. The bilateral movements of the eyes are rarely paralyzed, but individual muscles. The trigeminus is occasionally the seat of obstinate neuralgic pains. Corneal sensibility is usually diminished, whereas the cutaneous distribution of the nerve is rarely affected. Disturbances of a trophic nature are sometimes associated with the trigeminus affection; falling of teeth (Oppenheim), spontaneous fracture of alveolar process, lachrymation, hemiatrophy of the face have been described. Marie has described atrophy of the masseters. The acoustic is also occasionally affected, causing deafness, tinnitus or neuralgic pains in the canal. The hypoglossal nerve sometimes becomes affected, and hemiatrophy of the tongue results.

The affections of the vagoglossopharyngeus are so intimately associated with the crises that they will be considered together. The crises belong to the visceral disturbances of tabes, and consist in paroxysmal cramp-like affections of the viscera, with or without pain. The most common are gastric crises, which occur as sudden spasmodic contractions of the stomach, causing nausea or vomiting. They usually appear without the least warning, have no attributable cause, last from a few hours to many days, and then disappear as suddenly as they came. During the attack very little can be retained by the stomach, and sudden alarming loss in weight is sometimes seen. During the intervals between the attacks the stomach behavior is usually quite normal, and digestion unimpaired. If the attack is severe hematemesis may result. Premonitory symptoms, such as hallucinations of taste and smell, epigastric paresthesias, or pain in the neck, have been described. Intestinal crises occur, but they are more rare, much less painful, and are characterized by sudden uncontrollable evacuations of large mushy stools. Laryngeal crises are much less frequent than gastric crises. They are really attacks of spasmodic coughing accompanied by a distressing sense of tickling and sometimes by other vague crises, such as tachycardia, dyspnea, and sense of oppression over the precordial region. Vesical and rectal crises, i. e., painful spasms of bladder and rectum with sudden emptying of their contents, are also an occasional manifestation of the disorder. Pharyngeal crises have also been reported (Oppenheim, Bechterew).

The trophic disturbances of tabes consist of muscular atrophy, arthropathies, ulcerations, alteration of skin and its appendages, and vasomotor disturbances. The atrophy of tabes may be due to simple wasting from disuse, wasting due to disease of the peripheral nerves, or it may result from disease of the anterior horn cells. The isolated atrophies, hemiatrophy of the tongue, atrophy of a single muscle, are very likely neuritic in origin, but one or both limbs are often affected in the same way. It is to be recognized by the hard, dense sensation the palpated muscle offers to the touch, and resembles other forms of neuritis.

Atrophy of the central origin has been described and confirmed by histologic examination by Déjerine, Shaffer, and one of us. As Déjerine rightly says, it resembles a combination of tabes and chronic anterior poliomyelitis.

In connection with the atrophies of the lower limbs, Joffroy has described an interesting deformity of the foot called "Pied Bot," which is a pronounced pes cavus in which the toes are flexed, the arch of the foot much accentuated, and the long axis is greatly shortened.

The commonest and most striking trophic disturbances are the arthropathies. They were first described in their proper relation to the disease by Charcot. They have been found in almost every joint in the body, but occur most frequently in the knees. Suddenly and without any warning the joint tissues begin to swell. The parts become hard, nonfluctuating, painless, as a rule, without temperature, and often reach an enormous size. As the condition progresses the cartilaginous portions are also affected, and then the head of the bone. These are then absorbed, and the rough ends of the bones, being left in apposition, can be distinctly felt grating on movement. In other words, subluxation occurs. The knees and ankles are most often affected, but such arthropathy has also been described in the arms, shoulders, hips, mandible and vertebral column as well. The arthropathies are frequently accompanied by abnormal brittleness of the long bones, and spontaneous fracture may occur. Not all the arthropathies are as severe as the above description, however, and a form frequently occurs which yields to treatment and disappears completely. Trauma very often plays an important part in the production of these lesions, but usually no history of this can be obtained. Sometimes the reactions about the fragments of a fracture are so violent as to produce a condition similar to an arthropathy. We have seen this in one of our cases, a tabetic who developed an arthropathy of the ankle joint following an operation on a perforating ulcer of the sole. During his convalescence he struck the back of the right hand a sharp blow, felt no immediate discomfort, but noticed two days afterward a swelling on the back of the hand the size of a large walnut. Examination showed he had fractured the third metacarpal bone in its middle third. Strange to say, he made a complete recovery, the fragments uniting perfectly. Usually, however, the fragments unite with the production of enormous callosities and deformities.

The cutaneous trophic disturbances are represented chiefly by the perforating ulcer, "mal perforant" of the French. It is found usually in the plantar surface at the metatarsophalangeal articulation, but sometimes attacks the hands. The French have also described a perforating ulcer of the buccal cavity. It is always quite painless, usually begins with the formation of a vesicle, which soon breaks down and leaves a round, punched-out, dry ulcer. It progresses inward if left alone, involving bones and perforating the foot completely. Other cutaneous disturbances are local edemas, falling of hair, nails, etc., erythemas, and purpuric spots.

TREATMENT

Success in the treatment of locomotor ataxia has kept pace with the development of knowledge of the nature and cause of the disease. Though usually regarded as incurable, persistent methodical treatment does more to stay the development of the pathological process and to prolong the time of the victim's usefulness than in any other organic systemic disease of the nervous system. Haphazard, casual, unmethodical treatment should have no place in the handling of tabes. The treatment of the disease may for convenience sake be referred to under five heads:

1. Prophylactic treatment: The treatment of the attributed factors of its causation.
2. The treatment of the morbid process forming its anatomical basis.
3. The treatment of the distressing symptoms which the disease causes.
4. The treatment which has for its aim the reëducation of the extremities; the reëstablishment of certain physiological conditions.
5. The general systemic treatment, and plan of treatment.

Causal Therapy. The Advisability of Antisyphilitic Treatment.——Syphilis is the only causative factor of tabes that will be considered here, as the other attributed factors are discussed under general hygiene. Neurologists have not agreed as to the advisability of giving antisyphilitic treatment in cases of tabes. Some, like Charcot for example, have steadfastly held that such treatment is useless in every case of true tabes, no matter how clear a history of previous luetic infection the patient gives or how indifferently he may have been treated during the active period of the syphilitic poison, providing, of course, that the symptom-complex of tabes did not develop within a short time after the syphilitic infection, from two to four years, when the lesion might legitimately be considered a true syphilitic and not a parasyphilitic one. On the other hand, Erb and many others who have colossal experience with this disease teach that every case of tabes giving a syphilitic history should be put through a rigorous course of antisyphilitic medication, combined with general restorative treatment, if there are no apparent objections to such procedure, and providing the patient has not already received such treatment at the hands of another physician. No hard-and-fast directions can be given for the guidance of the beginner who faces this question for the first time. It need scarcely be said that it is of the utmost importance to adopt at once the most orthodox antisyphilitic treatment in every case of tabes in which there are true syphilitic manifestations of the skin, mucous membrane, bones, or nervous system, inflammatory or exudative.

Furthermore, the recent developments in the study of cerebrospinal fluids furnishes us important indications for the use of antisyphilitic remedies, and at present we are guided by this alone. On the other hand, if it is not possible to have the fluid competently examined, every patient who develops tabes within 10 years after the initial lesion, or who shows unmistakable evidences of an active progress in the disease, should be immediately subjected to vigorous treatment. These evidences are: persistent pains, extension of pains to new areas, development of crises, or rapid development of paralytic phenomena. A high cell count of the cerebrospinal fluid and excess in globulin mean activity in tissue lesions, and they should be treated in the same way until this disappears. The time is too soon as yet to say whether a cell count that has yielded to treatment and approached the normal limits may increase at a later period. At all events, it is as well to control these cases by lumbar puncture at stated intervals, and treat them accordingly if the cell count rises. It is just as well to disregard the Wassermann reaction in the serum or fluid, since it may persist even after the most vigorous treatment.

The selection of the remedy has to a certain extent been modified by the addition of Ehrlich's salvarsan to our pharmacal armamentarium. But the time is not yet ripe for the final opinion as to the relative merits of salvarsan and mercury. It is our belief at this time that the value of salvarsan is far greater than mercury in the treatment of all syphilitic diseases of the nervous system, including tabes. It is best administered intravenously, and at first a full dose, 0.6 gm., may be given; or it may be given in half doses, 0.3 gm., once a week for three or four doses. The treatment must be repeated frequently. The mistake that has been made in reference to salvarsan is that reliance has been put in a few doses. No result of a permanent nature need be expected until after several doses have been given. Our experience with neosalvarsan is still too limited to allow an expression of opinion as to its advantages over salvarsan, but we are prejudiced in its favor. Its advantages are the readiness with which it may be administered and the rapidity with which the dose may be repeated. We have given salvarsan freely in all cases where antiluetic measures were necessary, and have experienced none of the bad results published elsewhere. If strict surgical cleanliness is adhered to, care taken in neutralization, and freshly distilled water used, there should be no unpleasant sequelæ and very little discomfort to the patient. Care should be taken in introducing the needle into the vein selected, since injury to the opposite wall causes infiltration in the surrounding tissues, and in some cases a mild but painful phlebitis follows. In the latter event the arm, in case the median basilic is selected, should be kept perfectly quiet and ice applied along the swollen vein.

If it is decided to put the patient upon a mercurial course of treatment, our experience has been that the best results are obtained by inunctions. If this method cannot be adopted, it may be given hypodermatically. When it is decided to give the patient a course of mercury, one should enter upon it in no half-hearted way. From gr. xxx to xc of blue ointment should be rubbed in daily, each rubbing lasting at least from twenty to thirty minutes, and the course kept up from four to six weeks. If hypodermatic injections are given, they should be administered either as bichlorid of mercury, beginning with gr. 1-20 and increasing until either gr. 1-4 or gr. 1-2 are given every day, or salicylate of mercury suspended in liquid albolene in doses of one to two grains once a week. In either case it had best be given deep in the gluteal muscles, and high enough not to interfere with the patient's sitting. We have found that the best way is to have the patient lie prone and then, grasping the buttock firmly with one hand, plunge the needle, which should be at least one and a half inches long, to the hilt.

The greatest watchfulness should be kept over the condition of the patient's alimentary tract, skin, and weight. He cannot be kept too clean, too much in the open air, nor be too carefully fed. If one mistake more than another is made in putting a tabic patient through a course of mercury, it is that the physician is careless of the patient's loss of weight. If this cannot be controlled, the mercury should be stopped at once. After the patient has gone through the mercury treatment, he should receive a vigorous tonic plan of treatment for several months.

Treatment Directed Against the Morbid Process.—Innumerable measures have been suggested to counteract the progress of the morbid conditions forming the basis of locomotor ataxia. The majority of these have been found by experience to be nearly or quite worthless. On the original recognition of tabes by Duchenne, this clinician and many of his contemporaries waxed enthusiastic over the usefulness of the faradic current, while Remak, on the other hand, upheld the greater efficacy of the galvanic current. Wunderlich declared that nitrate of silver possessed greater power to check the sclerosis of the posterior column than any other medicine. His experience was corroborated in a measure by many contemporaries and followers, and until a few years ago it was one of the most widely known and used drugs in the treatment of tabes, although in truth it is of comparatively little use. Drugs that have received far greater credit than they deserve in the treatment of tabes are ergot, belladonna, arsenic, phosphorus, gold, aluminium, and platinum. Whatever virtue they possess, apparently or actually, is due to improvement of general nutrition, the blood and its nutritive derivatives which their use for a brief period may produce, or to their symptomatic effect. The truth is that there are no substances which experience has shown to have any effect in delaying the disintegration of the sensory neuron, although iodid of potassium is still very popular. The most common experience that we have is to find that patients with tabes have been treated by giving them great quantities of iodid of potassium, often in large doses. We desire to say emphatically that we have never seen anything but injury result from such therapy, and to deprecate its use. The iodids are not antisyphilitic agencies in the true sense of the term. They may, indeed and often do, facilitate the dispersal of a syphilitic lesion when it is of an exudative nature, but never when it is of a degenerative nature primarily. We have never seen the slightest benefit result from the administration of ergot, which, on the recommendation of Charcot and Hammond, achieved a reputation wholly undeserved. It should never be given. Strychnin and the glycerophosphates are extensively and deservedly used, but not with any view to influence the anatomical lesion of the disease, save by improving the general nutrition.

This part of the chapter would not be complete without a reference to serum therapy. It is well known that a few years ago Brown-Séquard startled the professional and lay public by his claims for the rejuvenating qualities of extract of the testicle. He and his followers were able to furnish many plausible reasons, comparative and physiological, for the use of this extract and analogous ones made from the brain, the spinal cord, etc. These reasons still hold good. The utilization of the extracts, known to contain substances resembling alkaloids having indefinite physiological action, has not been attended by sufficient success to warrant us in recommending any of them in the treatment of tabes.

Symptomatic Treatment.—In meeting the indications of the third caption, the physician will have abundant opportunity to display his therapeutic resources. The pressing claims are the relief of the lancinating pains. These may be so severe and so unyielding to every form of therapy that they demand the administration of opium or one of its alkaloids, but this should in every instance be kept as a last resource. Usually the pains can be ameliorated by the use of the coal-tar derivatives, such as phenacetin, antipyrin, acetanilid, or by combinations of these with alkalies, such as antifebrin, and by counter-irritation over the spine, such as by the actual cautery applied very lightly from the nape of the neck to the lower lumbar region, by spinal stretching and suspension, electricity, warm baths, and the application of pungent soothing medicaments to the skin.

The combinations of the analgesics which we find most serviceable are:

℞	Caffein. salicylatis	gr. i	(0.065 gm.)
	Phenol. salicylatis	gr. xv	(1.0 gm.)
	Phenacetin	gr. x	(0.60 gm.)

One powder to be given every two hours until pain is relieved.

If the pain occurs at night, and the caffein seems to increase the wakefulness, we employ the following prescription:

℞	Phenacetin	gr. x.	(0.60 gm.)

S. Dissolve in hot water and administer conjointly with chloralamid, in powder or elixir form.

Among the newer remedies pyramidon in doses of from 1½ to 5 grains is sometimes efficacious; dionin, gr. 1-3, has also been recommended.

In a similar way the analgesics may be combined with sulphonal or trional. Occasionally the pain can be relieved by the prolonged warm bath (temperature 98° to 102° F.), lasting from fifteen minutes to half an hour, and general faradization of the extremities. Rarely, wrapping the legs or thighs in flannel wrung out of hot water in which capsicum has been dissolved, or moistened with chloroform and ether, is of service. I have never seen any benefit to the pain from massage, mechanical vibration, or percussion of the nerve trunks, nor from the application of a spray of ethyl chlorid to the vertebral column. On the other hand, I have seen material benefit attend the application of dry cups to the spine, the use of the actual cautery, and of stretching. When all other measures fail to relieve, it becomes necessary to benumb the sensorium by the use of opium. In full knowledge of the danger that the patient is in who receives morphin for the relief of pain which is sure to return, the physician is nevertheless under moral obligation to his patient to make use of this measure in certain cases; but he who leaves a syringe with the patient or with one of the family to be used when the pain is unbearable, outrages the privilege conferred on him by the Hippocratic oath. Even though the patient may live in the country far removed from his physician, no shadow of justification exists for making him a morphin habitué.

Treatment of the various crises that sometimes occur in tabes usually demands the temporary use of morphin, and the fact that the stomach is disordered in its functions in the most common of these crises makes it necessary that it be used hypodermatically. There is no danger of the formation of the morphin habit, for just as soon as the crisis is over there is no further indication for its use. If the gastric crisis is of only slight intensity, a temporizing measure of some satisfactoriness is the oxalate of cerium, given in from gr. iij to x doses, in the form of either a pill or wafer. Its efficacy may be materially enhanced by combining it with gr. 1-6 of cocain. During and following gastric crises of tabes there are marked deviation of the degree of acidity of the stomach and falling off of the peptogenic properties of its secretions; and one must be guided by the condition of hyper- or hypoacidity which is present in the vomit in reaching a decision as to medication and alimentation at this time. While the crises last the patient must be fed by nutrient enemata. In gastric crises considerable relief is sometimes had by the intermittent application of ice over the stomach, spraying the epigastric region with chlorid of ethyl, and by touching the skin of the epigastrium with a glowing iron. Prolonged faradization of the abdominal wall has likewise seemed to me of service in a few instances. Vesical crises demand the administration of morphin to allay the overpowering distress in the beginning. After this the patient can usually be kept in a comfortable state, until the crisis ceases, by the giving of a mixture of chloral hydrate, fluid extract of belladonna, and fluid extract of hydrastis. Laryngeal crises frequently require inhalation of chloroform, but never up to the point of complete narcosis. As in other crises, the two most reliable measures are morphin and absolute quiet.

Injections of stovain, novocain, and fibrolysin into the spinal canal have been recently recommended by Pope, Secord, L'Hermitte, and others. Pope saw wonderful improvement after four injections of fibrolysin. The reflexes returned, gait improved, pains and paresthesia disappeared, and the patients were in every way astonishingly better! L'Hermitte saw a similar result. It would be interesting to hear how these patients were a year after treatment. Suffice it to say the method has not found favor as yet.

Long after the active progress of the disease has been checked, the lightning pains and crises may reappear at irregular intervals. In many instances this is probably the result of toxic irritating substances emanating from the intestinal tract or the liver. They are best treated from the start by vigorous catharsis and some of the so-called intestinal antiseptics. At the onset of the attack we prefer to give an ounce of castor oil, and then keep the patient on a low, non-fermentative diet. Intestinal or gastric crises will often improve astonishingly on ten-drop doses of castor oil combined with five grains of salol, and repeated every two hours.

Retention and incontinence of urine frequently call for special medication and handling, aside from the direct mechanical treatment, such as regular catheterization, washing out the bladder with sterile water, or water to which some alkali or antiseptic has been added. Some preparation of belladonna or hyoscyamus with fluid extract of hydrastis canadensis or ergot may be given internally with good results, and, naturally, urotropin must be given freely. At the same time the bladder should be galvanized through the abdominal walls in the following way: One large electrode, 6 by 12 cm., should be placed above the symphysis, and the other electrode of half the size and with a concavity so that it fits up close beneath the pubic arch, then a current of from ten to twenty milliampères allowed to flow through from three to five minutes. In some cases the mixed current, the galvanofaradic, seems to act more satisfactorily than the galvanic current alone. I have often found this expedient of considerable service both temporary and permanent. When the incontinence of urine becomes complete, it is necessary for the patient to wear a rubber urinal, and to have the bladder washed out once a day.

Surgical measures for the relief of pains and gastric crises have been employed with somewhat uncertain success. The older abdominal operations were always unsuccessful, and usually left the patients worse off than before. Within the last few years there has been great activity in intraspinal surgery, and intradural incision of the posterior roots corresponding to the painful areas has been done. It is a dangerous procedure, however, and should only be undertaken as a measure of last resort. In the first place, they are not always successful, fully half the number have had no relief, and, besides, we are taxing an already weakened organism. It is well known that tabetic patients do not withstand operations as well as other individuals.

Tabetic amaurosis is one of the saddest, and fortunately one of the more infrequent manifestations of tabes that call for individual medication in addition to that undertaken for the amelioration of the disease itself. A most astonishing occurrence, and one which cannot be explained, is that all tabetic manifestations occasionally cease when the amaurosis becomes complete. There is no measure that can be depended upon to influence the amaurosis, yet occasionally the injection of sulphate of strychnin does good, and it should in every instance be tried, beginning with gr. 1-100 and increasing it every day until the physiological action is plainly manifest. Recently the use of the double cyanid of gold and potassium has been recommended in the following solution:

℞	Cyanid of gold and potassium...............	20 cgm.
	Distilled water	10 gm.

Five drops of this solution are injected into the muscles of the back once a day, and each day the dose is increased by one drop until fifteen drops are given. Then it is stopped for a few days, or is given in reducing quantities until a dose of five drops is reached. In our hands this has been a failure in every instance. Other measures that have been recommended, such as the application of electricity directly to the eye, and mercurial inunctions, have no efficacy. Iodid of potassium should never be given in these cases, for it is unquestionable that such administration hastens the process in the optic nerves. In this connection we desire to say that we do not consider an impending tabetic amaurosis a contraindication to the use of salvarsan.

In the terminal stage of tabes there is great liability to the formation of bedsores over those parts of the body that have been the seat of continual pressure, and all possible care should be taken to maintain the nutrition of the skin and subcutaneous tissue of these parts, as it is very much easier to prevent their occurrence than to cure them. Attention directed to the texture, evenness, and covering of the mattress; a daily cleansing bath and frequent sponging with cold water and alcohol; attention to the state of the bowels and bladder, will usually prevent the occurrence of bedsores. If they occur despite these, they must be treated according to the requirements of modern aseptic surgery.

It is not necessary to speak in detail of the treatment of such conditions as perforating ulcer, tabic arthropathy, and the osteopathies that may occur. In addition to the general treatment of tabes, they require the same surgical and orthopedic measures that trophic troubles of different origin demand. Immobilization of the joint is the essential thing. The perforating ulcer is often extremely resistant to all forms of treatment, and occasionally it progresses to such a degree and is so associated with adjacent profound arthropathy that it requires amputation; but this, fortunately, is very exceptional. Hyperextension of the knees also occasionally calls for orthopedic appliances.

General Treatment.—In latter years the measures that physicians have come to rely upon more and more in the treatment of tabes are those that may be included under the head of physical treatment, including hydrotherapy, balneotherapy, electrotherapy, massage, purposeful movements, suspension, and rest.

As in most other chronic diseases of the nervous system, hydrotherapy is a valuable agent in tabes to improve the patient's nutrition and to maintain his strength. The special hydriatic procedure that should be used in a given case depends largely on the patient, his idiosyncrasies, and his reaction to water at different degrees of temperature, but not a little on the symptomatic variety of the disease also. It is of far greater service in the cases attended by marked hypotonia than in the sensory forms. The usefulness of the warm full bath to relieve the shooting pains and the muscular soreness following an accession of the pains has already been spoken of. If given oftener than three times a week, it has a relaxing effect which should be avoided. In many cases, and especially those in which the pain is not very severe, the half-bath, temperature from 85° to 75° F., of from two to five minutes' duration, with friction, given every day, is followed by a general gain in bodily vigor, renewed feeling of well-being, and improvement of nutrition. When it seems advisable to get cutaneous stimulation or irritation, salt, pine-needle extract, or a stream of carbon dioxid gas may be added to the water. As a rule, however, very little is gained by these procedures. The thermal element is the important factor, and to this are owing the good effects of a sojourn at many watering places. Strong, full-blooded patients who react promptly and with pleasant subjective sensations to the application of cold water, often find much benefit from the use of water of 70° to 65° F., given from the hollow hand of an attendant, accompanied and followed by vigorous friction, and from the use of a tonic bath according to the following formula: Hot-box until mild perspiration results; Charcot douche, temperature 90° F., reduced daily from two to five degrees until 65° F. is reached, pressure ten to twenty pounds, duration thirty to sixty seconds, applied to the back, chest, abdomen, and calves, and followed by a Fleury spray, temperature 65° to 75° F., pressure fifteen to twenty pounds, duration fifteen seconds, followed by light friction all over the body for from two to five minutes, depending on the patient's reaction, and a brisk walk in the open air.

When it is impossible to send the patient to a hydriatic institute, this procedure may be replaced by wrapping the patient in a dry, hot blanket for from ten to thirty minutes, giving him a hot drink, water, weak tea, or milk, if his digestive apparatus is in good condition; then when the cutaneous circulation, especially that of the extremities, shows the effects of this internal and external heat, water is forcibly thrown from a dipper upon the spine and over the abdomen and chest, or the patient is flagellated briskly and quickly with the ends of a towel dripping with cold water, and followed by friction.

Urogenital symptoms are often benefited by the use of cool sitz baths, temperature 75° F., duration two to five minutes. Many patients object to them because of the idea that it increases the pain, but nevertheless such a bath is often serviceable in helping to relieve the urinary incontinence. Some writers recommend for the relief of pain and for its general tonic properties the use of a cold wet pack, which, of course, becomes a warm pack after it has been in apposition with the body for a short time. It is said that the uniform warmth thus induced tends to mitigate pain and dissipate paresthesia. I have not seen much benefit from it.

Many patients with tabes are greatly improved by a sojourn of a few weeks, once or twice a year, at the thermal mineral springs of this country, and the rôle of balneotherapeutics (mineral water treatment in contradistinction to hydrotherapeutics, the external or internal use of plain water) in the treatment of tabes is an assured and an important one. In this country the hot springs of Virginia and Richfield Springs are the most important. In Germany, those of Oeynhausen and Nauheim; and in France, those of Lamalou and Balaruc are in best repute. The manner and method of using the warm salt baths are very important, but usually it is necessary, when patients are recommended to visit a certain spring, to leave this matter to the physician of the baths. Nowadays it is almost unknown for a patient to take a course of waters at any of the springs without first putting himself in the care of one of the many physicians who are to be found there.

Tabetic patients are also often benefited, especially if they are anemic, dyspeptic, and inclined to cachexia, by a short visit to one of the many medicinal springs in this country and in Europe, such as Poland Springs, Ragatz, St. Moritz. The regulation of diet and of exercise, the open-air existence, and the devotion of a proper number of hours to sleep, which are the usual entailments of such places, all help to improve the patient's nutrition, to husband his energies, and to increase his strength.

The Use of Electricity in Tabes.—Almost from the time when tabes was first recognized as an individual disease, electricity has been accorded an important place in its treatment. Duchenne and Remak set the example in Europe, and they soon had innumerable followers all over the civilized world. Electricity has been praised out of all proportion to its real efficacy by some, while others seem to take a malign pleasure in denying it any virtue, either in influencing the lesion of the disease or in mitigating its symptoms. It is quite impossible to estimate accurately what service it really renders in this direction, but it matters not whether its usefulness is due to suggestion or to some influence that it has in counteracting the process of decay in the posterior columns of the cord; so long as it helps to prolong the patient's life and makes it more livable it is deserving of employment. Electricity is utilized in tabes in the shape of the galvanic current applied directly to the spine; the galvanic and faradic currents to the peripheral parts, including the cervical sympathetic nerves, and as static electricity. Of all the procedures, galvanization of the spine is the most important. Many modes of applying it have been recommended. The two following methods are quite satisfactory: the negative pole connected with a large electrode (six inches square) is placed on the chest, and the positive pole connected with a smaller electrode (one to two inches square) on the spine, and moved slowly from the cervical to the sacral region, while a current of from six to ten milliampères is allowed to flow, the duration of the treatment being about ten minutes. This should be done daily, and in very few cases it is more satisfactory if the electricity is applied twice a day, each séance being of from five to ten minutes' duration. It is highly probable that the beneficial effect of electricity thus applied is commensurate improvement of the circulation of blood and lymph through the posterior columns, nerve roots, and adjacent tissue. The other method is to place the cathode firmly over the superior cervical ganglion at the angle of the lower jaw, the anode over the opposite side of the spinal column, close to the spinous processes, and allow a current of four milliampères to pass. The positive pole is then rubbed up and down the spine for about five minutes on one side, then the cathode is changed to the cervical ganglion of the other side, and the same procedure used for the opposite half of the spinal column. In this way the posterior roots and the intervertebral ganglia are stimulated, and, in view of the important part taken by disease of these structures in the pathogenesis of tabes, it can readily be seen that this is a desirable operation.

Some amelioration of the paresthesia may confidently be expected from the use of faradic electricity applied to the skin of the extremities. Greatest excitation of the cutaneous nerves is obtained by using the small brush electrode. If it is desired to stimulate the peripheral neuromuscular apparatus, either the faradic or the galvanic current may be employed. When it is elected to use the latter, the positive pole should be used as the differentiating electrode. I have seen some benefit follow the use of static electricity, applied by means of a large brass ball electrode or by a roller. Numerous theories have been suggested to explain this beneficial action, but its use is based entirely upon empiricism. Some maintain that the sensory nerves conduct the electric energy along their course to the cord, and the current thus exercises a beneficial action upon the entire sensory neuron. More plausible is the explanation that the sharp muscular contraction caused by the spark improves the neuromuscular tone. It need scarcely be said that electrical treatment should not be relied upon exclusively. On the contrary, it should be looked upon as an adjuvant of some importance, and given in connection with other physical and medicinal treatment. No more is it advisable to give electricity month after month without interruption. Its effects seem to be best when it is given for a period of six weeks three or four times a year.

In certain cases of tabes the regular, persistent use of massage does a great deal of good. At least it gives more comfort than almost any other measure. It is especially useful in cases of long duration. It counteracts muscular hypotonia and asthenia in a more gratifying way than any other measure, and I have often seen the symptom described as "giving way of the knees" entirely disappear under this form of treatment. Women tolerate massage and benefit from it far more than men, and this rule holds good in tabes. The most useful massage procedure is stroking. Diligently and carefully used to the muscles of the back, it overcomes the dorsolumbar hyperesthesia, and, applied to the extremities, it not infrequently assuages the pain and mitigates the hypotonia. Vigorous kneading and compression of the back often decrease the girdle sensation, while general massage may be used for its tonic effects. Stretching of the peripheral nerves either by operation or by the bloodless method, which formerly had considerable vogue, is to be severely frowned at. No doubt such procedure sometimes relieves pain, but the same results can be obtained by having the patient lie on the back with the head slightly elevated and the legs extended; an attendant then grasps the feet and draws them back toward the patient's head, the knees remaining extended. This position is maintained for from two to four minutes, and repeated once or twice a day.

Suspension a Therapeutic Agent of Some Value.—Suspension seems to have been first recommended by Motschutkowsky in 1883. In the decade following neurologists of every nationality testified to its efficacy in ameliorating the symptoms of tabes, and apparently in modifying the course of the disease. During the past few years very little has been heard of it. Nevertheless it is still in use, and a dispassionate estimate of its value based on experience would probably hold that its methodical use is attended by a lessening of the severity and frequency of the pain, by an improvement in walking, by some amelioration of the vesical symptoms, and by an improvement in the patient's general morale. The contraindications for suspension are cardiovascular lesions, pulmonary tuberculosis, emphysema, apoplectic and epileptic attacks, a tendency to syncope, and obesity. It should never be used in the paralytic stage of tabes. The patient may be suspended in the Sayre apparatus, but the most expedient way is to suspend him from the head and elbows while seated by means of a specially constructed harness which hangs from one end of a horizontal beam that is movable pivotally on a firm perpendicular. Weights are attached to the other end of the horizontal beam, and by increasing or decreasing the number of weights the desired degree of suspension force can be had. Its *modus operandi* is not very clear, but it probably acts mechanically to stretch the spinal column and its attachments, and thus indirectly to improve the circulation in the spinal vessels.

Reëducation of the Ataxic Extremities, Fraenkel's Method.—Difficulty of locomotion eventually becomes the most conspicuous burden of the patient's life. So long as he is able to get about unaided he may live not only a useful, but comparatively an enjoyable, life; but, when he has to rely upon the arm of an attendant, a pair of crutches, or a wheel-chair, fortitude deserts him, and with it hope and usefulness.

Of all the measures that can be utilized to counteract the development of ataxia, and to overcome it after it has developed, the plan suggested by Mortimer Granville of England in 1881, but formulated and introduced to the profession by Fraenkel of Heiden in 1890, and since then very much elaborated by himself and by Goldscheider, is the most important. The essential feature of the plan is to submit those muscles which manifest the incoördination to a series of graduated and systematic exercises. Each movement thus performed will be accompanied by kinesthetic sensations and memories in the corresponding areas of the brain. Fraenkel has, therefore, referred to the treatment as one of cerebral gymnastics, and in no way to be confounded with gymnastics of force. The underlying principle is that, if the patient is made to overcome the ataxia by the performance of simple movements with purposeful intent and attention, his sensorimotor cortex will become so reëducated that it will direct the movements without attention and conscious volition.

Fraenkel's procedure consists essentially in the exact and methodical execution of purposeful movements which require skill and not force. In the beginning these movements should be very simple, and gradually made more complicated as the patient becomes capable of performing them. They should not be done in a perfunctory way, as are ordinary gymnastics, but with the attention closely concentrated on every movement.

The benefit which follows the use of the exercises is often most encouraging to the patient and gratifying to the physician, especially when used as an adjuvant to the general tonic and supportive treatment already spoken of. Naturally they are of signal service in those cases in which the ataxia and hypotonia are not extreme. Oftentimes, however, patients who are dependent upon crutches may be so benefited by this treatment that they can walk unaided, particularly if the hypotonia is not profound. It must not, however, be forgotten that these exercises have no influence on the course of the disease, and that they benefit only one of the numerous clinical manifestations, namely, the ataxia.

The cases of tabes most favorable to the employment of Fraenkel's treatment are those in which the ataxia appears very early in the disease; those in which it is of comparatively slow development; and those in which the incoördination manifests some tendency toward spontaneous amelioration. The employment of this method of treatment is contraindicated in weak, anemic patients and in those who suffer more or less constantly with pains or crises; in cases of acute and subacute tabes, that is, in cases of sudden onset and in which the habitual manifestations of the preataxic period succeed each other rapidly; in patients with tabid optic atrophy, fragile bones, and those who have had what is generally called spontaneous fracture or rupture of tendons. When any cardiopathy or vascular lesion exists, the method must be tried with great care, if at all. It is not applicable to very obese or arthritic patients, and, finally, it is absolutely contraindicated when there are great laxity of the ligaments and severe arthropathy.

The formula for these exercises, which was given by Fraenkel in his communication to the Moscow Congress in 1897, seems rather formidable. The following résumé will aim to give the more important essentials. The illustrations are taken from Goldscheider's brochure on the subject. The physician who undertakes to employ the exercises must needs remember that they demand for their successful utilization a great deal of time and patience, but expenditure of them will be rewarded. Whenever it is possible the exercises should be entrusted to a trained attendant under the observation of the physician. At the Neurological Institute, where we utilize them to great advantage, they are always done in classes and to the accompaniment of music. They should be practiced several times a day for a few minutes at a time, but never to the point of producing considerable fatigue. When they cause great fatigue they should be done principally in the morning or after a long rest.

Ataxia of the lower extremities is commoner and always more severe than of the upper; it is also more difficult to overcome by the Fraenkel movements, because of the associated disorder of equilibrium, which is often so profound. To overcome the ataxia of the legs the patient should begin by making simple, primitive movements that can be executed while lying in bed. For instance, lying on the back with the legs uncovered, he should be required to go through movements of flexion, extension, abduction, and adduction of the different joints of the lower extremities, slowly and deliberately and with all the accuracy that can be commanded, first with one leg, then with the other, and finally with both legs simultaneously. Fig. 1 illustrates one of the simplest and most important of these movements. It is spoken of as the "fourfold movement exercise." It consists in raising of the outstretched leg, flexion of the thigh, and then the knee, to make a double right angle, then extending the leg, and finally lowering it. It will be found that these simple exercises are very fatiguing, not so much because of the muscular exertion, but because of the attention that they demand. They should be persisted in, however, until the patient can execute them easily, accurately, and without much effort. Another very important series of exercises is represented by Fig. 2. As seen from the illustration, it consists of a short step ladder fixed at the bottom of the bed on which the patient is required to make accurate stepping and climbing movements. Similar movements of precision should then be practiced by the patient while sitting. It is unnecessary to detail the great number of modifications of such movements that can be devised. Care and precision in their execution are most important. These primitive movements are absolutely essential and should not be neglected even by those whose ataxia is not so great as to prevent them from walking.

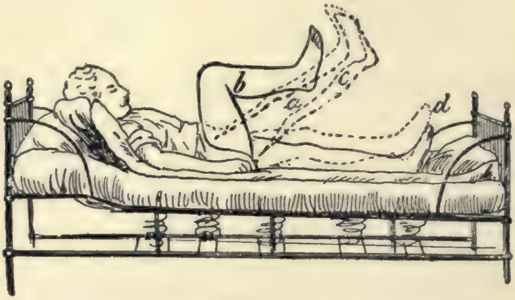

FIG. 1

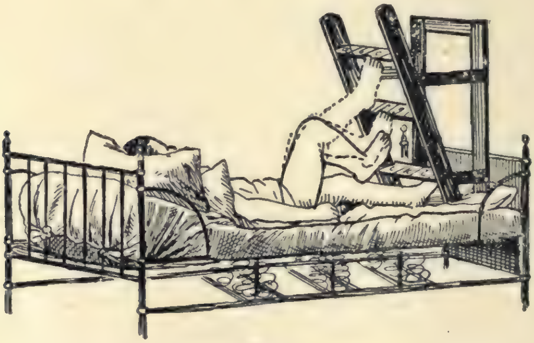

FIG. 2

The patient should then practice rising deliberately from the sitting position with or without aid, as the condition of his strength and equilibrium demands, and then sitting slowly. As soon as possible he should do this without assistance or support. He should then practice standing upright alone or with support or assistance, with the feet put firmly beneath him, aided at first by the hand or eye, then, as he gradually acquires confidence and skill, without the aid of either and with the feet close together. The physician can be of great service by assuring the patient of his capacity to do this, for much of the disequilibrium is the result of fear and lack of confidence. When he has succeeded in learning to stand alone or with the aid of a stick, he should begin movements of bending forward as far as possible, then slowly raising himself to a vertical position, bending first one knee and then the other, adopting the squatting position and then rising from it, and various others.

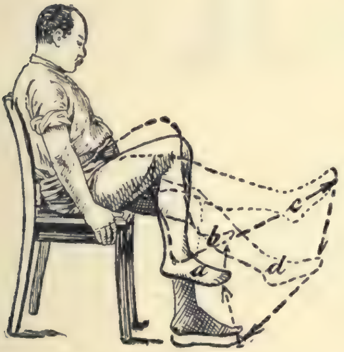

FIG. 3

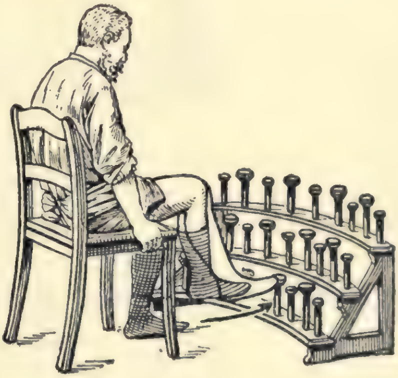

FIG. 4

After this movements of the lower extremities looking toward walking are to be practiced. The patient should stand with the aid of crutches or a stick and endeavor to put one foot forward slowly, deliberately, and accurately upon a certain marked spot, from twelve to sixteen inches in front of him. This should be done five times in succession with all possible precision and accuracy, while the patient or the attendant counts. Then the same procedure is to be repeated with the other foot. After the patient has acquired facility in doing this he should try to walk with the aid of sticks or an attendant, taking each step slowly and with the greatest deliberateness, ten steps forward, then ten steps backward. The patient should also be required to practice walking movements of the feet while sitting. This should be done first with one foot, then with both feet simultaneously, as shown by the accompanying illustration

(Fig. 3). Another exercise of considerable service is illustrated by the simple apparatus (Fig. 4). The patient first touches all the round-top shorter uprights and then all the flat-top taller uprights in succession.

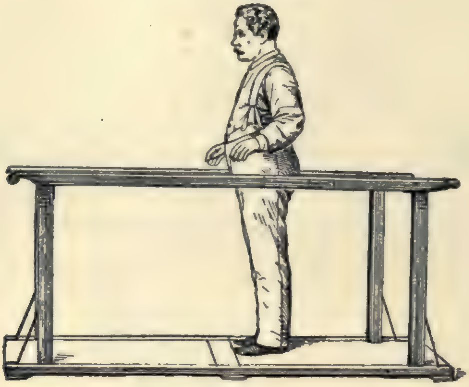

FIG. 5

Of course, when the patient cannot stand or walk even with the aid of a stick or an assistant, it is necessary to provide him with some such apparatus as shown in Fig. 5, by which he can support himself while practicing the many different exercises of skill. The exercises that can be devised with such a contrivance are almost innumerable. A few of the important ones are illustrated by Figs. 6 and 7.

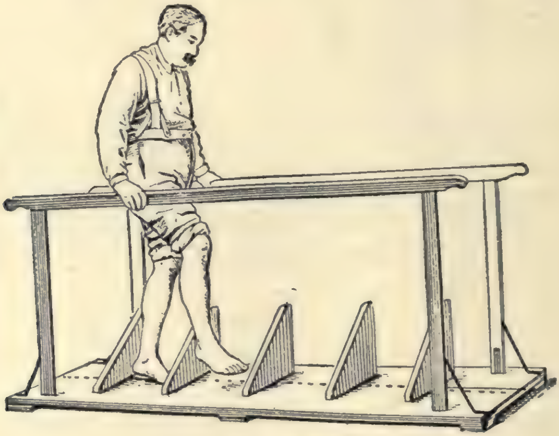

FIG. 6

By assiduous practice of these exercises the patient will soon be able to walk without holding an immovable support. It then remains for him to practice walking in a straight line; to trace figures or lines with the toes and to indulge in other movements of the lower extremities that require accuracy and skill for their execution.

To overcome the ataxia of the upper extremities one proceeds in a similar fashion. The patient is made to execute simple movements at first, followed by more complicated ones, with the fingers, hands, and forearms. When the patient can use his muscles without difficulty for these movements, he should be given exercises which require more skill and patience for their performance. Fraenkel has devised for this purpose a number of apparatuses. One of these consists of a piece of wood having the form of a triangular prism forty centimeters long, each side measuring five centimeters. This piece of wood rests on one of its sides. The upper edge is grooved out, one of the others is smoothed off, while the third is sharp. This apparatus is placed before the patient, who holds in one hand a large pencil, and he endeavors to put the point of the pencil in the groove and move it back and forth therein steadily and accurately from the farthest to the nearest end, keeping the fingers and the wrist immovable. At first the patient has considerable difficulty in keeping the point of the pencil in the groove, but after repeated exercises he is able to do so. He should then practice retracing with a pencil simple designs consisting of straight, zigzag, and curved lines. If the tracings of these figures are kept, it enables one to follow the progress that the patient makes toward acquiring coördination. Another apparatus consists of a piece of board in which depressions have been hollowed out at regular intervals, into which the end of the finger can be placed. These holes are numbered. The board is placed before the patient, who holds the right arm raised and the index finger extended.

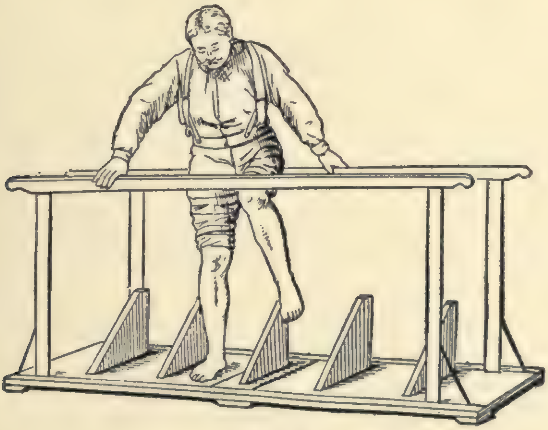

FIG. 7

He then puts the end of the finger into the depression rapidly and as accurately as possible when the attendant calls out the number. At first he is made to repeat the same number until he can do it with considerable accuracy. This exercise can be made more complicated by having the patient put marbles in the holes as fast as the number of the holes is called out. This simple device may be replaced by a board filled with holes, in which the patient is required to place a number of pegs, such as a cribbage board.

The great number of variations that can be devised by any one, even the patient himself, will suggest themselves at once to the physician. After the patient has acquired skill in these exercises he can practice with a contrivance consisting of a series of balls of different sizes suspended by threads from a horizontal bar. One of these balls, commencing with the largest, is made to oscillate, and while it is oscillating the patient is told to seize it and bring it to a state of rest. At first he is left free to choose the moment at which to seize the ball, but later he is required to do so at a moment determined by its oscillatory excursion. This exercise is repeated with all the different balls. The smaller the ball the more difficult the exercise.

O. Foerster has described a definite series of exercises for the upper extremities. They should be done serially, as they are designed to proceed from simple movements and attitudes to the more complex ones. They are as follows:

1. Resting each finger and thumb in the normal attitude of rest.
2. Resting all fingers and thumbs at the same time in the normal attitude of rest.
3. Slow extension and flexion of the fingers at the metacarpophalangeal joints, at first separately, then together.
4. Holding the second and third phalanges in any given position of flexion, at first separately, then together.
5. Synchronous extension and flexion of the second and third phalanges.
6. Extension of the end and middle phalanges while the proximal is flexed.
7. Apposition of the thumb to the fingers.
8. Crooking the thumb and first finger so that the balls touch.
9. Approximation of the extended thumb and first finger so that the balls touch.
10. Holding quiet all the fingers and thumb while the volar sides of the corresponding fingers at the last phalanges are approximated.
11. Separating and bringing together the finger balls of the individual pairs while the hands are held as in No. 10.
12. Grasping a can and piling up several.

13. Grasping the pointers and placing them in the holes of a cribbage board.

14. Placing the fingers in the grooves of a special board and slowly raising and sinking them.

15. Practice in buttoning and unbuttoning.

16. Holding the hand quietly in the normal position.

17. Slow extension and flexion of the fingers.

18. Opening and closing the fist.

19. Grasping larger articles with the hand.

20. Shaking hands.

21. Holding the hand and forearm in a given position, then deliberate pronation and supination.

22. Holding the forearm in a given position of flexion, then deliberate flexion and extension.

23. Holding the uplifted arm in any given position of the shoulder joint, then deliberate raising and lowering.

24. Seizing large objects with the whole hand.

25. Seizing swinging balls of the Fraenkel apparatus.

26. Carrying a spoon to the mouth.

27. Touching the nose with the finger tips.

28. Practice in writing.

29. Copying figures, lines, etc.

30. Keeping the pencil in the groove of Fraenkel's triangular block.

Plan of Treatment.—The importance of a plan of treatment for patients with locomotor ataxia can scarcely be overestimated. The medicinal treatment will not suffice unless supplemented by hygienic, dietetic, physical, and disciplinary measures. The amelioration of the patient's symptoms and the degree to which comfort and longevity can be given him stand in direct relation to the promptitude with which the diagnosis is made and proper treatment instituted. Every patient who consults a physician for an ailment of any kind, aside from the acute infectious diseases, and who gives a history of syphilis, should be carefully put through the tests requisite to reveal the existence of tabes. The necessity of this statement is impressed upon us as our experience with the disease increases. The patients who have been treated for rheumatism, gout, appendicitis and who have been operated for malignant disease, stricture of the urethra or rectum, spinal cord tumor, and other diseases "too numerous to mention" testify to the truth of this. It is scarcely necessary to emphasize how essential it is closely to scrutinize the patient who comes complaining of pains, vesical shortcomings, altered genesic instincts and capacity, or any of the symptoms of the general neurasthenic condition. At such an early date it may be quite impossible to satisfy one's self of the existence of tabes, but if there is an increase in number of the lymphocytes in the cerebrospinal fluid, it is imperative that the patient be given salvarsan or mercury *properly and adequately.*

When the existence of the disease is unmistakable, the question must always be decided whether or not to inform the patient. Naturally no general rule can be given, but it is my own belief that nothing whatever is to be gained by concealing from the patient the nature of his disease, for fear that such knowledge may have an injuriously depressing effect if he has heard of the disastrous outcome of the disease in others. If the first physician whom the patient consults does not apprise him or one of the family, which is tantamount to telling the patient himself, of the nature of his disease, it is very likely that sooner or later he will consult another physician who sees his way clear to impart such information. The first physician will then be held to have made a mistake and to have wasted time in treatment which might have been beneficially employed if the exact nature of the disease had been known. It is not wise in the majority of cases to make an unmodified diagnosis of locomotor ataxia in the beginning; nevertheless the patient should be made to understand that his disease is serious, and, in order that it may not disable him, it is necessary to adopt a vigorous plan of treatment for the purpose of stopping the progress of the disease. The important lesson for him to learn is that a long period of usefulness and comparative health waits on methodical and continued treatment. Occasional and desultory visits to a physician which are rewarded by one or two prescriptions are tantamount to no treatment at all.

After getting *en rapport* with the patient and securing at least a degree of his confidence, it is necessary to decide whether or not he shall forego his customary occupation, providing, of course, his position in life allows him to do so. Individual factors in each case must influence this decision. As a rule, unless special indications exist to the contrary, such as a profound neurasthenic state, manifestations of syphilis in the blood vessels, progressive emaciation, and unless the occupation is one that is conducive to leg weariness and entails great worry and care, it is best to let the patient keep to business. His infirmity prevents him from indulging in many of the pleasures and occupations which help to pass the time. And to take a man accustomed during all the years of his life to engrossing occupation, and throw him at once into enforced idleness, at the same time restricting him from many pleasures which are harmless to the healthy individual, is tantamount to converting him at once into an introspective, depressed, miserable being. On the other hand, if he is allowed to pass a portion of his time in business, while the rest is given over to measures that may be legitimately called treatment, such as walking, golfing, driving, not to speak of the time required for hydriatics, electricity, massage, and rest, he will have little time to think of himself. A rule that admits of few exceptions is that sanatorium treatment is not advisable, at least not until the last stages of the disease.

Tabetics do not tolerate brusque changes of temperature or oscillations of atmospheric pressure. Such changes are apt to be accompanied or followed by attacks of pain, gastric crises, exaggeration of ataxia, and general asthenia. A temperate climate and a moderately dry atmosphere are most favorable for patients with this disease.

In regard to the patient's diet, it may be said that tea, coffee, alcoholic stimulants, and tobacco should be used most temperately. It is poor judgment to insist that a man who has taken these dietetic luxuries in moderation and to his apparent benefit for many years shall give them over entirely, just because certain nerve fibers are beginning to decay. A mixed diet with a preponderance of vegetables and fats is the nearest approach to the ideal. As in all nervous diseases, functional or organic, the patient should be fed frequently, five times a day at least. The supplementary meals should consist of milk or other food, the taking of which requires no effort on the part of the patient. The tabid patient who spends twelve out of the twenty-four hours in bed is more just to himself than he who encroaches upon this number. In regard to exercise very little can be said in a formal way. Leg weariness is to be avoided at all hazards. It will be found that the varieties of exercise and sport that are in vogue in any country or section of the country for the average healthy man can be indulged in quite as well by the tabid patient, providing he is not inclined to be intemperate. It is unnecessary to speak of the importance of avoiding injurious indulgences of all kinds, but patients in the early stages of tabes often seek advice concerning attempts at sexual intercourse, for in many instances sexual potency is not entirely lost until the disease is quite advanced. So far as possible, cohabitation should be avoided, although if there is no pathological excitation no harm can result from occasional indulgence. The general hygiene, including clothing, cleansing baths, regulation of bowels, maintenance of the integrity of digestion, does not require specific mention. As in all other nervous diseases, attention to these details is rewarded by an accession of the patient's nutrition and increase of physical strength.

The use of drugs to maintain nutrition constitutes relatively an unimportant part of the treatment of tabes. Of the general tonics and restoratives iron, arsenic, and quinin are occasionally serviceable, while the simple bitters and dilute hydrochloric acid are to be used for their direct effects on the appetite and digestion. There would seem to be a fixed conviction on the part of many practitioners that strychnin is a drug of surpassing value in all diseases of the spinal cord. In reality it is not, and in tabes its unguarded use may be followed by increase of the lightning pains and irritation in the urogenital sphere. A few years ago we gave it a thorough trial, most of the patients being at the end of the second and beginning of the third stage and hospitalized. It was given hypodermatically, beginning with one-sixtieth of a grain and gradually increased to one-sixth of a grain per diem. Its proclivity to cause pain was guarded against by the simultaneous use of small doses of morphin. No discernible benefit followed its use. It may be used as a tonic, but specific effect should not be anticipated from it.

FRIEDREICH'S ATAXIA AND HEREDITARY CEREBELLAR ATAXIA

Hereditary spinal ataxia is a degeneration or lack of development of the peripheral sensory neuron and the central motor neuron in their spinal course, constituting posterior and lateral sclerosis of the cord. It is a rare disease of childhood, very chronic in its course, and unamenable to every form of therapy. The three important etiological factors of the disease are: the family history, the age when the symptoms first occur, and the relationship of acute disease. The name hereditary spinal ataxia is misleading, because in at least one-third of the cases there is no evidence whatever of immediate or remote heritage of the disease, and in upward of ten per cent. of the cases there is no history of pathological heritage of any kind.

The most striking fact in the etiology is the occurrence of the disease in more than one member of the family, though even this is not discernible in all cases. It is more apt to occur in large than in small families, and at times it seems to affect the male members while the females escape, and vice versa. Although all the members of a family are not affected, unless in exceptional instances in which the number is very small, the remaining members may show some other form of degenerative nervous disease, and possibly nervous disease of a teratological nature. The immediate and remote family history may show the existence of some degenerative neurosis or psychosis, such as epilepsy, hysteria, inebriety, and migraine. The disease develops, as a rule, between the ages of five and fifteen; it sometimes occurs in a recognizable form before that period, and has been recognized as early as three years; the number of cases occurring after the fifteenth year is not very great, and they probably belong to the cerebellar type.

It has often been noted that, when the disease occurs in several members of the same family, it appears in the first patient within late childhood or early maturity, while in each succeeding patient it appears at a less advanced age. The factors that apparently have something to do with exciting the disease, at least to such activity that it becomes recognizable, are the infectious diseases—naturally those common to childhood—and injuries. The influence which these factors have may be interpreted in two ways: The acute infectious diseases may have nothing whatever to do with causing the disease except in so far as they weaken the neuromuscular system and keep the patient in bed, during which time complete coördinated movements, such as walking, running, and climbing, which the person may have but recently mastered, are partially forgotten. Either of these factors, or both combined, may be sufficient to make noticeable the most striking feature of the disease, viz., incoördination, which had existed before the infection. On the other hand, infectious processes and their products may act injuriously upon neurons robbed by heritage of their complementary development and cause them to degenerate. This latter belief I hold to be extremely improbable. A number of cases have been reported in which the disease was ushered in by febrility. What the genesis of this fever is has not been suggested, but it seems that the explanation of its injuriousness is the same as that offered for the infectious diseases. A number of other etiological factors of comparatively insignificant importance are the occurrence of the disease more frequently in males than in females, oftener among the poor than among the rich, and the recording of no case in other races than the white. These facts, with the exception of the last named, are in entire accord with the teachings of other familial and hereditary diseases, all of which show themselves more frequently in males and in people of the lower walks of life. The disease is met with in the poor and unenlightened, because parental consanguinity, excessive fetation, and malnutrition are commoner.

Symptomatology.—The most striking feature of Friedreich's disease is the disturbance of gait. It consists of a profound disturbance of equilibrium; ataxia in all purposeful movements of gait and static incoördination, and in well-developed cases there is a play of jerky, inconstant muscular movements for the purpose of maintaining equilibrium. The ataxia during the earlier appearances of the disease may be limited to a slight unsteadiness of gait, or awkwardness of the hands and arms on attempting finely coördinated movements. When the incoördination is very pronounced, all sorts of equivalent postures are adopted to maintain equilibrium, i. e., tilting of the pelvis, forward stretching of neck and head, and lateral balancing movements of the trunk. The purposeful muscular movements are executed with the same irregular, exaggerated joint excursions as in tabes. Athetoid and choreic movements have also been described. Muscular weakness is a very prominent feature in the later stages, and is accompanied by more or less symmetrical wasting. Actual paralysis is rare, except when the joint movements are limited by the deformities. Muscle tonus is in no way lowered, as in tabes, and is usually increased. Romberg's sign is not always present, and in Friedreich's original article it is described as absent. It has since then been observed many times, and its presence or absence has no particular diagnostic significance. The reflexes are either diminished or absent. Sometimes an apparently absent knee or ankle jerk may be elicited on reinforcement. Diminished myotatic irritability usually accompanies the muscular wasting. Babinski's sign is nearly always present, whereas the cutaneous reflexes conform to no general rule. Deformities occur in very many cases. They appear in the spine, usually as scoliosis, but often marked lordosis is seen. Perhaps the most constant deformity is that found in the feet. It consists in a well marked pes cavus, Friedreich's foot or pied bot. We consider it to be a sign of great diagnostic importance. In addition to the shortened high arch, there is a peculiar extension of the proximal joint of the great toe with flexion of the distal joint. Claw hand has also been described (Menant). Nystagmus very frequently occurs in these cases, sometimes static, sometimes dynamic. It is not always constant, however, and when it occurs we should always suspect the cerebellar type of the disease. The usual behavior of the pupils is normal, although Argyll-Robertson pupils have been noted. Optic nerves are also normal, but we have seen one case, that of a young girl of twelve, with typical Friedreich's ataxia, in which there were double optic atrophy and sluggish pupils. Another cerebral feature of the disease is the peculiar slow, jerky, at times explosive, nasal speech disturbance. The voice is monotone, and there is often a definite catch of breathing between words or even syllables. It is perhaps the most striking instance of ataxic speech that we encounter.

Sensory disturbances do not occur, as a rule; but paresthesias, hypoesthesia of the extremities, diminished position sense, and lancinating pains have been described. The ordinary trophic disturbances of tabes are practically never found. Bladder and rectal functions are rarely disturbed, although the sedentary life these patients lead usually induces constipation.

Hereditary cerebellar ataxia (ataxie-hérédo-cérébelleuse) was described by Pierre Marie in 1893, and has since been brought into direct connection with Friedreich's ataxia by later writers. The general belief is that they are different clinical pictures of the same fundamental process, and that they differ rather in the distribution of the lesions than as clinical entities. Hereditary cerebellar ataxia appears usually at a later age than Friedreich's, i. e., 20 years, and is more rapidly progressive. In a few instances it has been known to appear as late as 45 years. The etiology is as obscure as Friedreich's, but its family and hereditary features are more constant. One can nearly always distinguish the familial manifestations, but it is not always so easy to establish the hereditary traits. Males are more often affected than females. The same etiologic features prevail as in Friedreich's disease, and, just as the latter depends upon developmental anomalies in the cord, so it depends on faulty structural development of the cerebellum. Therefore, it is our belief that injuries, infections, emotional disturbances can have significance as contributory factors only.

SYMPTOMS.—The symptoms of hereditary ataxia are: uncertain, reeling, staggering, titubating gait, in which there is often a distinct spastic element. The French justly compare the gait to that of an intoxicated individual. The patient walks with the feet wide apart, pelvis forward, trunk backward, the head backward and swaying from side to side. The equilibrium is maintained in the sitting posture or on lying down, but the ataxia persists on purposeful movements during both these postures. There is marked exaggeration of mimic during speech or emotions, which is not at all similar to the emotional play in multiple sclerosis or lesions of the basal ganglia or tegmentum. The speech resembles that of Friedreich's ataxia, but is more explosive and jerky. The tendon jerks are always exaggerated, ankle clonus is sometimes present, and the Babinski phenomenon is always to be found. Coarse, irregular nystagmus is frequently, although not invariably, present, and sometimes paresis of the external recti. A very constant feature is the mental impairment which sooner or later appears during the course of the disease. It varies from slight stupidity to dementia; idiocy, epilepsy, and Menière's syndrome have been described as possible complications.

Pathologic Anatomy.—The spinal cord is remarkably thinned in these cases of Friedreich's disease, and the cerebellum often is also remarkably small. This is regarded by most writers as a definite anomaly of development. In the Marie form only the cerebellum shows this defect. In Friedreich's disease the degeneration involves principally the posterior columns and direct cerebellar tracts. The lateral pyramidal tracts are, as a rule, moderately affected; the direct pyramidal tracts are untouched. Clark's columns are severely affected, but the anterior horns and the spinal roots, both anterior and posterior, are usually unaffected. The cerebellum is atrophied to a remarkable degree in Marie's type. It is a general atrophy of all the elements. The arborizations are small, the central white masses are thin, and appear like lamellæ. The cellular elements are usually but not always affected. Diminution in size and number of the purkinje cells has been noted. In both types there is astonishingly little secondary reaction in the glia, small round cells, or in the vessels. The cerebellar connections with the oblongata, i. e., the restiform body and pons, are usually smaller, and the tracts degenerated.

Diagnosis.—The differentiation of these two forms is often difficult, and sometimes impossible. Exaggerated reflexes suggest the cerebellar type, pes cavus suggests Friedreich's type. Exaggerated emotional facial play suggests Marie's type, as do mental disturbances. On the other hand, deformities suggest Friedreich's type or mixed forms. It is sometimes hard to distinguish these types from multiple sclerosis. Often it is impossible to do so, but the absence of abdominal reflexes, the irregular course of the disease, attended by progression and retrogression and accompanied by emotional disturbances, are in favor of the latter. A steady, slow progression with the characteristic deformities is in favor of Friedreich's disease.

Treatment.—The treatment of these two forms of ataxia is identical and consists in providing the patient with an intelligent attendant or nurse who will practice and instruct him in the system of purposeful gymnastics, known as Fraenkel's movements. This system of systematic exercises for training the ataxic limbs is described in the chapter on the treatment of tabes. These, with measures taken to maintain the strength and nutrition of the patient, are all that can be offered in the shape of therapy. It is not probable, even though we treat the patient from the very beginning of the disease, that medicines, such as silver, aluminium, and iodid of potassium, which sometimes have a beneficial effect in preventing the rapidity of development of certain spinal cord degenerations, would be of any service in this disease. The spinal curvature rarely calls for direct treatment, but many patients are more comfortable when they wear a light wooden or plaster jacket. Parents to whom are born one or more children who afterward manifest a disease of this kind should be advised to surcease procreation. If they do not, the endeavor should be made to avoid the factors that seem at times to act as exciting causes—the infectious diseases and injuries.

Spastic hereditary ataxia, spastic heredodegeneration, or hereditary ataxic paraplegia has been described. These cases really belong to the mixed forms of the group just described, and are characterized by the prominence of the spasticity, exaggerated tendon jerks and the more or less rapid course of the disease.

ACUTE MYELITIS

The term myelitis has been and is applied both clinically and pathologically with much latitude. It is used to indicate the changes in the cord, the result of acute inflammation, disease of the walls, and partial or complete obliteration of the lumen of the vessels (myelomalacia), pressure upon the substance of the cord the result of accident, disease or new growth of the surrounding tissue, and the vascular and parenchymatous changes developing from lessened atmospheric pressure (caisson disease).

The designation acute myelitis should be restricted to indicate an acute exudative and destructive inflammation of the spinal cord involving the white and gray matter, of variable extent, in vertical or transverse direction, and occurring at any level. As a rule, the inflammation is of the dorsal or upper lumbar segment, and the focus of the morbid process is more extensive in a transverse direction. Thus the disease is often spoken of as acute transverse myelitis. The pathological products vary with the inflammatory excitant and with the intensity of the infection. The trend of modern scientific thought is to associate the occurrence of inflammation with some bacterial cause, but there is nothing approaching unanimity as to what constitutes the essentials of inflammation or inflammatory reaction. It is quite impossible to distinguish clinically the myelitis which is the result of a pathogenic organism, such as that of influenza or typhoid fever, from the myelitis or myelomalacia that accompanies syphilitic degeneration and thrombus of some of the spinal blood vessels. Neither is it always possible to distinguish them anatomically, after the cases come to autopsy, for the exudative and destructive changes that go on around such a focus or a number of foci are practically identical with those of primary inflammation. Indeed, the reactionary changes around such foci may be so great that they more or less obscure them and prevent their ocular demonstration. The pathological product of acute myelitis is seldom pus, except in those rare instances in which the myelitis is due to pyogenic organisms, in which case it may be circumscribed to constitute a more or less diffuse abscess of the spinal cord. Purulent myelitis is almost invariably associated with and secondary to purulent leptomeningitis.

Acute myelitis may be classified *regionally* with respect to its location in the cervical, dorsal, or lumbar regions; *topographically* according to major extension as transverse and longitudinal; *etiologically* as traumatic, infectious, toxic, and refrigerant; and *clinically* as acute and chronic. When the inflammation of the cord is accompanied by or is secondary to inflammation of the meninges it is known as meningomyelitis.

The Etiology of Acute Myelitis.—The causes of acute myelitis are the same as those of other acute parenchymatous inflammations. Naturally, certain influences are more harmful to the spinal cord than they are to other tissues. Any depreciation of the circulation and nutrition of the cord, or, in other words, any diminution of its resistivity, may act as a powerful predisposing cause to microbic invasion. In this way is to be explained the action of cold, fatigue, especially of the legs, such as is induced by prolonged or violent muscular effort, sexual excess, and trauma insufficient to cause solution of continuity. These factors are usually considered exciting causes of acute myelitis, and very frequently some one of them is the sole detectable cause. Of these attributed causes exposure to cold is by far the commonest and most pernicious. It is possible that of itself it is sufficient to excite inflammation in the cord, as this has been done artificially in the lower animals by means of an ether spray. Infection is more liable to occur in middle adult life than at any other age. Despite the fact that acute myelitis often develops in the wake of infectious diseases, children are rarely affected. There is no preferential liability with respect to sex other than that engendered by the occupation of males, predisposing by exposure, fatigue, the action of poisons, and the liability to injury, and by pregnancy and the puerperal period in the female. The insignificant seasonal relationship of the disease, viz., its more common occurrence in winter and spring, is clearly related to exposure and cold.

The infections that are most frequently followed by acute myelitis are: pneumonia, typhoid fever, erysipelas, diphtheria, influenza, puerperal fever, malaria, gonorrhea, infectious endocarditis, scarlatina, and variola. Of these the infections of pneumonia and influenza are by far the most pernicious. How these infectious agencies act to produce myelitis is not clearly understood. Naturally, their direct presence in the spinal cord would be certain to set up inflammation. But it is much more probable that they produce poisons of the nature of toxins which single out the spinal cord for their pernicious activity. In this way is explained the occurrence of myelitis some time after the infectious disease with which it stands in causal relationship has ceased to exist. Whether or not the immediate pathological precedent of such infectious myelitis is a minute embolus or thrombus has not been definitely decided, but it would seem that, in some cases, at least, this constitutes the first pathogenic step. Acute myelitis has been produced experimentally in animals by the injection of cultures of erysipelas bacilli, colon bacilli, staphylococci, pneumococci, tetanus bacilli, Loeffler's bacilli, Eberth's bacilli, etc. Of the infections, tuberculosis is probably the most common. It is usually but not invariably associated with involvement of the meninges. In a case studied recently we were able to demonstrate the tubercle bacillus in the myelitic area.

The poisons that stand in causal relationship to the occurrence of acute myelitis are of endogenous and exogenous origin. The latter are least important, although lead, arsenic, mercury, phosphorus, and carbon monoxid are occasional attributable causes. The rôle played by alcohol in the causation of acute myelitis is not a very prominent one. It acts indirectly by leading to exposure and injury, rather than by its inherent perniciousness on the cord. Toxic agencies arising within the body have a more malign influence. The most important of these are due to diabetes, uremia, gout, and rheumatism. Acute myelitis sometimes occurs with disease of the urinary organs, such as cystitis and pyelitis. An attempt has been made to explain such occurrences by saying that it was an extension of inflammation or of the inflammatory excitants directly from the tissues primarily diseased to the cord, but this is wholly unlikely, and the myelitis is probably due to injurious agencies circulating in the blood and having their origin from these diseases. The occurrence of myelitis with exfoliative dermatitis and after burns that have denuded a considerable surface of the body is explained in two ways: First, that these lesions allow the development of poisonous substances which are absorbed into the system; and, second, that they act upon the sympathetic system to produce vasomotor derangements in the cord which go on to inflammation similar to those resulting from cold.

Trauma is a relatively uncommon cause of myelitis, except in those instances in which the trauma is sufficient to produce physical disintegration of the substance of the cord, as from fracture and dislocation of a vertebra. Slighter trauma may open the surface to the invasion of bacteria, or it may cause marked depreciation of the circulation and nutrition of the cord.

Myelitis is met with in a number of blood diseases, such as profound anemia and leukemia, occurring primarily or secondarily to malignant disease, such as carcinoma, and to some chronic disease, such as nephritis. Here, again, it must be said that the lesions forming the anatomical basis of such forms of myelitis are not true inflammatory ones. Their pathogenesis consists in the occurrence of minute thrombi or emboli, with resulting myelomalacia, which cannot be distinguished from acute myelitis. The acute myelitis that occurs in animals when the blood supply is shut off by pressure upon or ligation of the aorta, and in man with aneurysm and partial occlusion of the abdominal aorta, is pathologically a true anemic necrosis, with subsequent surrounding reactionary myelitis.

Myelitis may be secondary to an inflammation of the surrounding structures—the meninges and the vertebræ—although this is not an important causation. There is some evidence tending to show that it may be secondary to an ascending peripheral neuritis, particularly from the nerves of the trunk. Such a case has never come under our own observation.

Pathology.—On removing the cord the meninges are usually injected, cerebrospinal fluid is increased, and the cord is softer than normal. On account of its consistency, artefacts are very easily produced in the removal, and they are often difficult to distinguish from the true lesions. If the lesion is an acute transverse one with intense inflammation, the consistence will be pulpy. In the acute stages the cut surface shows small reddish punctate areas, digitations pushing in toward the center, obliteration of the markings, small areas of necrosis, and occasionally cavities; the vessels are swollen, tortuous, and stand out prominently. In later stages the reddish areas are transformed into grayish-whitish patches, the necrosed areas are more easily distinguishable. The meninges are thickened, especially the piaarachnoid, or may not appear changed in any way. Where the process has proceeded from the meninges they are thickened, glued together, and sometimes the subdural space is filled with a glairy gelatinous mass. Histologically, we find lesions of the most varied degree. In severe transverse cases the markings of the cord disappear, the white and gray matter are indistinguishable, the nervous elements are no longer recognizable, except here and there a pale, poorly stained ganglion cell or a few swollen axones. The glia persists as a few indeterminate fibers. The area is composed of granular cells in various stages of necrosis, and a poorly staining amorphous mass of necrosed tissue. In the less severe types there are small foci of variable sizes, scattered irregularly throughout the diseased area. They are sometimes round, appearing in white or gray matter, or as digitations pushing in from the periphery of the cord. The foci are composed of granular cells of vascular, glial, or connective tissue origin; spider cells usually around the periphery of the focus, swollen axones, fragments of myelin, and occasionally endothelial cells. The reaction in the fibers is more or less intense. The axones are swollen and tortuous, the myelin fragmented, and the sheath swollen to two or three times its natural size. The swollen sheaths frequently become confluent with the edge of the focus, and form large spaces, which are called Lückenfelder by the Germans. Marchi stain shows intense degeneration, fatty infiltration of the granular cells, the vessels, and the glia. The vessels within the foci exhibit all forms of degeneration, i. e., hyalin, thickening of all three coats, emboli, thrombi, or endarteritis obliterans. The softening process may spread by confluence of several foci, or the latter may remain isolated. If the process is severe enough, the softened necrosed material becomes absorbed, and small cavities appear and enlarge. When the foci appear in the gray matter the ganglion cells within are destroyed or distorted, the nucleus often being the only element that preserves its tinctorial reaction to any degree. Those on the edge of the focus are in various stages of chromatolysis, from the coarse granular to the dusty appearances of the Nissl bodies, and there are usually increase of pigment and formation of fat droplets.

As the acute process subsides the necrotic elements are absorbed and the process of repair begins. This is accomplished by the glia, which begins to proliferate and form new fibers that hypertrophy and form the so-called scar formation. Those of the true nerve fibers that have not been completely destroyed become clothed with the myelin sheath again. The optic nerves when affected are sometimes simply swollen and edematous, but usually show small foci similar to those in the cord. The foci usually contain small hemorrhages or transudations.

The purulent forms are rare, and usually occur with meningomyelitic lesions. The pus infiltrates the arachnoidal meshes, everywhere there is leukocytic infiltration, and the direct extension from the vertebral column can usually be demonstrated.

Symptoms.—The symptoms of acute myelitis vary with the location and extent of the lesion. The introductory symptoms, which are independent of the location of the inflammatory foci, may come on with great abruptness, constituting the apoplectic variety, or in a few days, constituting the acute variety, or in a few weeks and often somewhat intermittently, constituting the subacute variety. Usually the first sensory and motor irritative symptoms are followed by more or less complete paraplegia. When the lesion is of the dorsal cord, its commonest location, the symptoms consist of paraplegia, pain in the back radiating into the trunk and legs, and more or less anesthesia, paralysis of the bladder and eventually of the rectum; exaggeration of the knee jerks and later spasmodic twitchings and contractions of the leg; vasomotor and trophic disturbances, consisting of bedsores, slight edema of the legs, coldness of the extremities, and occasionally the formation of bullæ. The muscles waste, but do not atrophy, and there is no reaction of degeneration. When the inflammatory foci are in the lumbar region, the paraplegia that occurs is of the flaccid variety, and there is atrophy of the muscles with reaction of degeneration. The skin reflexes are weak, and the tendon reflexes are usually lost. There is a variable amount of anesthesia in the paralyzed parts, and the rectal and vesical insufficiency is profound. When the myelitis is of the cervical cord, the general symptoms are more profound and there is, in addition to the symptoms indicative of dorsal myelitis, motor paralysis of the arms or of individual muscle groups, usually of an atrophied character. There may likewise be oculopupillary symptoms, disturbance of respiration, and bradycardia. If the lesion is adjacent to the oblongata, the bulbar symptoms will be more pronounced.

Oppenheim described a conus type, in which the symptoms were paresis of the bladder and rectum, sexual impotence, anesthesia of perineum, anus, penis, scrotum, and the upper portions of the inner surfaces of the thighs.

The incomplete forms of myelitis exhibit, as one might imagine, a most variable group of symptoms. As the process is not necessarily limited to one segment, it may be distributed widely throughout the cord. In not a few cases the spinal symptoms are preceded by retrobulbar neuritis and by optic neuritis, whose origin is very puzzling. The optic affection may be limited to one or include both eyes. We have recently recorded two such examples. After such symptoms have existed for a short time, the real spinal affection appears, simulating tabes dorsalis, multiple sclerosis, or ataxic paraplegia. It is very complex in its clinical appearance, and often is only to be distinguished from the latter by its rapid course. In these forms we frequently see the Brown-Séquard type of dissociated sensory disturbances. If the infection is a severe one, it is accompanied by fever, chills, and sometimes delirium, stupor, and speech disturbances from the beginning. The onset may occasionally simulate acute poliomyelitis. In the subacute and chronic forms there may be an interval of months before the disease has reached its height. As yet there is little to be learned from a study of the cerebrospinal fluid in these cases.

The course of the disease varies with the causation and with the intensity of the infection. It is always progressive, but after a variable time, unless death occurs, it becomes more or less stationary, with resulting secondary degeneration, ascending in the sensory tracts and descending in the motor tracts, the latter predominating. The disease eventually causes death by exhaustion and by infection from the urinary organs and bedsores.

The diagnosis is made by the abrupt or rapid onset and establishment of the disease in a few hours, days, or weeks. It is to be differentiated principally from multiple sclerosis by its course, fever, and absence of emotional phenomena. The diagnosis of tumor compression from disease of the vertebra will be discussed later in its proper place. It is to be differentiated from embolus of the aorta by absence of pulsation in the iliac arteries.

The prognosis varies with the intensity of the infection. In severe transverse lesions it is unfavorable. In the disseminated types the chances of life are better, but complete recovery has been recorded only a few times.

The gonorrheal form is the most favorable, most of the cases recovering completely in a short while—6-12 weeks (Oppenheim). The cases occurring in puerperal fever frequently recover. Those during the menopause are rather unfavorable. It has been said that those cases with acute onset and multiple distribution of symptoms usually have a favorable prognosis, though this has not been our experience.

Treatment.—Considering the almost invariable outcome of acute myelitis, the treatment of the disease is thankless and dispiriting. Nevertheless, much can be accomplished by appropriate treatment to limit its extent, to alleviate suffering and misery, and to avoid in a measure some of the distressing secondary occurrences. The possibility of an abortive treatment of acute inflammation of any organ is problematical, but it is certain that there is none for an acute inflammation of the spinal cord. Yet something can be accomplished in the direction of lessening the intensity of the inflammation and shaping its course toward partial restitution. The appropriate treatment naturally varies with the cause of the disease, although all varieties of acute myelitis call therapeutically for two things: first, *absolute rest;* and, second, *absolute cleanliness.* It may legitimately be said that, just in proportion as these two requirements are fulfilled, so will the chances of partial recovery of the patient and the duration of life be increased. The patients should be put to bed and kept there, and they should not be allowed to move under any circumstances. The changes of position which are advisable, either to keep the parts on which pressure is most severe from becoming the seat of bedsores, or for the purpose of influencing the circulation in the cord, should be done by an attendant. It is advisable, if the condition of the patient allows it, to have him lie on the belly or side for a part of the time. The greatest care should be exercised in the selection of a mattress and in the arrangement of the coverings and clothing of the patient, so that irregular pressure on the surface of the body is avoided. Whenever it is at all possible, the patient should at once be put upon a water bed. Unfortunately, the physician sometimes waits for the occurrence of trophic symptoms before insisting upon this. Much trouble and suffering can be avoided by ordering it at the beginning. The most scrupulous cleanliness must be insisted upon. Warm water and soap should be used at least twice daily, followed by rubbing the skin with alcohol and by dusting with the blandest antiseptic powder. The condition of the bladder and bowels should be made an object of special attention from the start. If this is neglected, symptoms are sure to develop which point to infection, intoxication, and depreciation of vitality, and which will seriously jeopardize the patient's life. When it is impossible to catheterize the patient regularly, males should be provided with a urinal so adapted that every drop of urine passes into it, while females should have absorbent cotton surrounded by gauze or oakum so arranged that it catches every drop, and this should be renewed every two hours at least for the first few days, and after each renewal the parts thoroughly cleaned. Urotropin and other substances that have antifermentative properties should be administered freely. The bowels should be moved regularly by the use of simple enemas. If there is incontinence of feces, efforts to secure and maintain cleanliness must be redoubled.

If the myelitis is post-infectious, the treatment required, in addition to that mentioned above, consists in the administration of medicines that prompt the emunctories to activity, so that the elimination of the poison from the system may be facilitated. It is advisable to give an intestinal laxative and antiseptic, such as a dose of calomel followed by a saline, and a few brisk doses of some bland diuretic and diaphoretic, particularly if the patient is a robust, full-blooded individual, and to follow this by the administration of small doses of salicylates and quinin, both of which, fortunately, tend to alleviate the pain. If the case is seen in the beginning, it is very advisable to put an ice-bag over that portion of the spine where the lesion is situated, whenever an opportunity is offered by a favorable position of the patient. All forms of stimulant and irritant applications to the spine should be rigorously avoided during the acute stage of the disease. The skin is the seat of profound depreciation of nutrition, and it does not tolerate such irritation. The insignificant benefit to be derived from such applications is enormously disproportionate to the chances that are taken of causing or hastening decubitus. Of the drugs that have been recommended for their specific action upon the inflammatory process, two only—ergot and belladonna—need be mentioned, and these to be condemned. The use of ergot has been common for many years, but experienced physicians are gradually losing faith in it. We have never seen anything approximating benefit result from its use. The same may be said of belladonna, although the latter may exercise a stimulating effect on the bladder and be of some benefit. Pain should be relieved by the administration of phenacetin, combined with one of the salicylates, and by morphin, which should not, however, be given hypodermatically. Involuntary twitchings of the lower extremities are best controlled by the latter drug, but when they are not very severe they can be mitigated by the occasional administration of a dose of one of the bromids. The fact that this latter drug is a vasomotor depressant, however, should not be lost sight of.

When the myelitis is due to blood diseases, such as anemia and leukemia, in brief, when there are grounds for the belief that the myelitis is in reality a myelomalacia, with secondary inflammatory reaction, the treatment is somewhat different. In such cases the administration of eliminatives, the application of cold, and the giving of drugs that have any lowering influence upon the circulation are contraindicated. We can judge of the existence of these conditions only from the history of the patient and the accompanying manifestations. Such patients require supporting, stimulating, and alterative treatment from the beginning. Active antisyphilitic treatment is at times of signal service in cases in which there is a distinct syphilitic history, especially if the treatment is begun early and carried out vigorously, i. e., repeated salvarsan injections *and* the use of mercury. The treatment must not be carried out in the beginning to the exclusion of treatment looking toward the restitution of the blood vessels that are the seat of degenerative and exudative changes. The general treatment is the same as given above, but should include in addition small doses of cardiac stimulants, such as strophanthus and digitalis, combined with moderately increasing doses of iodid of potassium. If the myelitis is secondary to blood diseases, the treatment is the adoption of measures looking to the cure of the condition to which the myelitis is secondary, and the administration of substances that support the patient's vitality. The same may be said of myelitis occurring secondary to autointoxications. They are to be combated directly quite apart from the superadded occurrence of myelitis, but the latter is to be treated as well. It is unnecessary to enumerate the special indications of causal therapy in each one of these conditions.

In all cases care should be taken to brace the patient to withstand the onslaught upon his vitality and to maintain as far as possible the integrity of the peripheral circulation. The first is to be encompassed by careful administration of nutritious, easily digested food, given frequently and in small quantities, and if necessary by the administration of alcoholic stimulants in small quantities. The second can be accomplished in part by the application of dry heat to the lower extremities, by frequent and prolonged immersions of the extremities or the entire body in warm water, after which they are wrapped in cotton wool, and by the use of mild massage. It must again be mentioned that the vitality of the skin is such that it will resent rough handling of any kind, and care must be taken in the application of hot water bottles and in the use of manual friction.

Electricity has been recommended for its attributed efficacy in mitigating certain symptoms, such as incontinence of urine, for preventing muscular atrophy, and for its direct effect upon the spinal cord. It may be stated positively that it should never be used with any such end in view as specific action on the cord. In some cases it would seem that a large electrode connected with the positive pole and placed above the pubes over the bladder and the negative on some indifferent point, while a current of from two to three milliampères is allowed to flow, is of some service. After the acute stage has subsided, either the faradic or the galvanic current may be used to stimulate muscular contraction and especially to prevent inactive muscle atrophy. As a rule, it may be said that it is much safer not to use electricity during the acute stage.

In some cases, even in those in which the greatest care has been expended in carrying out the essential requirements in the treatment of every case of myelitis, viz., rest, cleanliness, frequent change of position, absolutely smooth surface to lie upon, tonifying measures, etc., untoward symptoms, such as cystitis, pyelitis, bedsores, and other trophic phenomena, occur which require particular and careful treatment. Such treatment, however, is not at variance with the treatment applicable to similar conditions developing under other circumstances. Cystitis occurring with myelitis requires for its successful treatment a careful study of the urine, the administration of substances that make it as bland and unirritating as possible, and the local or intravesical application of substances that combat the inflammation. Frequent and thorough irrigation with plain warm water, or, better still, with some simple alkali and antiseptic solution, such as a five per cent. solution of boracic acid, a two per cent. solution of salicylic acid, or an extremely weak solution of nitrate of silver (1:1,000), should be used two or three times daily. Vesical irrigations with carbolic acid and sublimate solution have been recommended, but their virtues are not sufficient to counterbalance the discomfort and danger attending their use. Pyelitis is to be treated according to general principles of rest, administration of large quantities of water, and small doses of salol or urotropin, with the same attention to the diet as indicated in the ordinary case of pyelitis. Bedsores are to be treated with antiseptic solutions and dressings the same as acute ulcers occurring in a debilitated subject. The danger in attempting to stimulate them to healthy reaction is great. When they cannot be controlled in this way, the patient must be put for a time in a continuous warm water bath.

After the acute stage of the disease has passed comes the time for the adoption of measures looking to the absorption of the inflammatory residue and the mitigation of the consequences of the injury. The nutrition of the patient should be carefully studied. It is not only necessary to administer appropriate food, but to get the patient into the fresh air by means of an invalid roller chair if he is unable to walk, to administer measures that contribute to sleep, overcome constipation, and to maintain nutrition of the muscles and the integrity of the peripheral circulation by massage, passive exercise, and as much active exercise as it is possible for the patient to take. It is at such times and later that regular cures should be undertaken, either at home or abroad, at thermal springs and health resorts, such as the Hot Springs of Arkansas and Virginia, Glenwood Springs, Colorado, Richfield Springs, New York, Lamalou, France, Nauheim and Oeynhausen, Germany, and such places as have obtained repute in the treatment of different varieties of degeneration of the spinal cord. A sojourn at one of these places frequently results in much greater benefit than can be explained by the taking of the waters internally or externally. It not infrequently improves the patients' morale; the observances there require the maintenance of great cleanliness, which in turn betters the peripheral circulation, and the disciplinary measures to which they are subject facilitate metabolism and increase the appetite. All of these are of the greatest importance. Many men experience a partial or temporary restoration of the sexual power from such treatment; this improvement benefits them by inspiring hope and imbuing confidence.

In cases of myelitis secondary to diathetic conditions this is the period when there is some hope of using constitutional and non-medicinal measures to great advantage. It is also the period when rigorous antisyphilitic treatment should be carried out if such treatment seems to be indicated, as it is in every case in which there is a syphilitic history, whether or not the patient has had what seems to have been adequate treatment following the infection.

As yet there is little to be obtained from serum therapy in most cases. Where a definite infectious agent is demonstrated as still active the vaccines or sera may be tried. In every case of myelitis of suspected gonorrheal origin the vaccines should be given. Thus far the results have been disappointing, but the trial of them has been wholly inadequate.

CHRONIC MYELITIS

Under the foregoing title the combined pseudosystem diseases of the cord will be discussed, as well as chronic myelitis proper. This has been made possible by the recent researches of Nonne, Henneberg, Mayer, and others, who have demonstrated that for the greatest part this group derives its origin from small foci in the various tracts. Nonne is inclined to confirm Leyden and doubts the existence of true combined system disease of the cord.

Etiology.—The causes of chronic myelitis are: (1) All the causes of acute myelitis, as the chronic variety may be one mode of termination of the acute; (2) syphilis, which is by all means the commonest single cause, it being found in at least one-third of all the cases, and has already been discussed; (3) poisons, such as ergot, which has the peculiarity of causing destruction, particularly of the posterior column, alcohol, lead, mercury; (4) autointoxications, gout, diabetes, and chronic anemia. The predisposing causes are practically the same as those of acute myelitis. Exposure to cold and wet is the attributed cause in many of them. Fatigue and prolonged physical activity and strain are noted in many others. The disease is likely to occur during the years of early maturity, and much oftener in males than in females.

A variety of chronic myelitis dependent upon senile changes in the spinal blood vessels, senile arteriosclerosis with resulting perivascular sclerosis, occurs occasionally in old age, and is known as senile paraplegia.

Of 40 consecutive cases diagnosticated as chronic myelitis, 32 were males and 8 females. The average age of the patients was thirty-seven years. Out-of-door manual laborers furnished 42 per cent. of the entire number. Fourteen of the 32 patients gave a history of syphilis, and in 11 of these the symptom-complex of myelitis conformed to the type known as syphilitic spinal paralysis. Thirty-three per cent. of the cases gave a history of exposure to cold, and in the majority of these the refrigeration was considered the cause of the disease by the patient. In 15 per cent. of the entire number the disease was secondary to acute myelitis, and in the majority of these there was a history of acute infection, such as influenza or pneumonia, or of exposure. In 8 per cent. of the cases there was a history of injury without evidence of its previous existence. One patient had diabetes and 2 suffered from severe and chronic anemia. Only 1 case was of the senile variety.

The symptoms of chronic myelitis which are sequential to the acute variety will depend very largely upon the severity of the original process. They are practically the same as those of acute myelitis, save that they are less profound. When chronic myelitis is chronic *ab initio,* as from exposure and exhaustion, the symptoms usually consist of: 1. Heaviness and easily induced fatigue of the legs; 2. Stiffness of the lower extremities in the beginning, particularly after arising and after resting, but later the stiffness is constant; 3. Exaggerated tendon reflexes, i. e., knee jerks, ankle clonus, Babinski and Oppenheim phenomena; 4. Urinary symptoms, particularly manifested in difficulty in emptying the bladder, later incontinence; 5. Impaired sexual capacity; 6. Variable and inconstant sensory symptoms, consisting of objective numbness of the legs and feet, tension around the lumbar and lower abdominal regions, and occasionally paresthesia of different parts of the lower extremities. The symptoms of the senile variety are a gradual development of a slightly spastic paraparesis associated with mild vesical symptoms. These symptoms become more pronounced and oftentimes the arms present analogous but less marked symptoms. In some cases arteriosclerotic changes in the brain, similar to those responsible for the senile paraplegia, produce the symptom-complex of senile dementia or other symptoms of encephalomalacia.

Treatment.—The treatment of chronic myelitis divides itself into treatment of the syphilitic cases and the non-syphilitic. In the former the amount and duration of antisyphilitic treatment which the patient will tolerate must be decided in each case, and this cannot be decided properly without study of the cerebrospinal fluid. Aside from this and the causal treatment of myelitis, mentioned in the discussion of the acute variety, the treatment consists in so arranging the patient's life that he is saved bodily and mental agitation and fatigue, that he is spared the injurious action of alcohol, tobacco, and narcotics, and that he is vouchsafed a life of intelligent rest and exercise. These, and the employment of agencies to meet the symptomatic conditions and measures to improve nutrition, constitute the entire treatment. As soon as the paraplegia reaches that degree of development that locomotion is difficult and fatiguing, the patient should be encouraged to get about in a roll-chair. Spasticity is to be combated by frequent warm baths of from ten to fifteen minutes' duration. Many patients receive benefit and much comfort by remaining in such a bath for an hour or even longer.

Electricity is of no service in influencing the course of the pathological process. If there is muscular atrophy, either from inactivity or of other origin, electricity may be used with some success to combat these conditions. But as a rule both the galvanic and the faradic current tend to increase the spasticity, and should not, therefore, be employed. Massage and passive exercises are much more useful. Massage not only improves the circulation and the nutrition of the parts, but, when combined with gymnastics, tends to preserve mobility and to facilitate voluntary movements.

Internal medication with the object of overcoming the lesion in the spinal cord is not very encouraging. Nitrate of silver has been recommended, but it is useless; ergot has been praised, but it is injurious. Phosphorus and arsenic have been given, but no one can say positively that they do any good; if they do, it is probably by improving the nutrition. Strychnin is a drug that is used extensively in this disease by the general practitioner. But, aside from its stimulating effect on the bladder, we have never seen any beneficial effects follow its use. By increasing the spasticity it may hamper the patient's locomotion and it may exaggerate the involuntary contractions, which are so annoying, especially at night, to many patients.

Local treatment over the spine, such as the application of the cautery, counterirritants, vesicants, etc., and especially the former, are sometimes of service. Such treatment seems to be quite as important for psychical as for physical effects.

The general health is frequently bettered by the employment of a mild cold water cure and by massage. They have a beneficial effect *per se,* and they likewise benefit by making the patient feel that something is being done for him. The symptomatic treatment is the same as in acute myelitis. The condition of the bladder and of the skin should be made objects of special solicitude.

Combined Sclerosis of the Spinal Cord, Combined Pseudosystem Disease, Funicular Myelitis

The various designations of this condition represent the somewhat different views of the investigators as to its cause. It is usually associated with pathologic blood states, such as pernicious anemia or chronic anemia.

Etiology.—The etiologic factor is most probably a toxin arising in the low state of vitality of the individual, and may proceed from various exhaustion-producing diseases. Lesion of the cord occurs frequently in pernicious anemia, and has been considered by some to result from the anemia, although its absence in severe hemorrhagic anemias and hemophilic states seems to argue against this view. We believe it should be regarded as the effect of the same toxins causing the pernicious anemia. It is found in the acute form, sometimes in leukemia, but here it usually appears as a more or less severe meningomyelitis with small foci of lymphocytes scattered throughout the cord and leptomeninges. Various other causes have been indicated, such as exposure to cold and wet, exhausting labor for a long period of years associated with chronic gastric or intestinal disorders, chronic alcoholism, and chronic nephritis, although in this instance the low-grade changes in the blood vessels are rather to be regarded as the cause. It is never found in the cachexias of tuberculosis or malignant diseases.

Pathology.—Macroscopically the cord may appear quite normal or only slightly shrunken, the cut surface showing small grayish sclerosed patches in the lateral pyramids and posterior columns. Microscopically we find small inflammatory foci in the ascending and descending tracts, usually the posterior columns, the lateral pyramidal tracts, direct cerebellar and Gowers' tracts, and sometimes the anterolateral ground bundles and the columns of Türck. It may be limited to small funiculi in the tracts, or may involve the entire tracts. There is almost invariably a zone of normal fibers around the gray matter, which also is usually intact. The meninges are only slightly affected, the roots never. The vessels may be nearly normal, or show hyalin degeneration, sclerosis, and thickening of all coats. The marginal portions of the cord are more severely affected.

Symptomatology.—The onset is often insidious, beginning with weakness of the legs, rigidity of the muscles, paresthesias, numbness, tingling, etc., and the disease may progress rapidly to complete disability within a few weeks, or it may take months to develop. We have seen one case in which the symptoms came on rapidly after direct transfusion, a procedure which had most remarkable effect upon an enemia considered primary. The symptoms will naturally depend upon the tracts affected most severely. The reflexes may be paradoxical, i. e., knee and ankle jerks absent and Babinski and Oppenheim phenomena present, all may be abolished or all may be exaggerated; extension of the big toe on stroking the sole of the foot is usually present. Sensory disturbances vary from light hypoesthesia to profound anesthesia of all qualities. Bladder and rectal functions are usually disturbed, but not always. There is always in the beginning some degree of ataxia, although it may consist merely in a slight unsteadiness of station. The eyes, pupils, and cranial nerves are unaffected.

The disease is recognized by the presence of a pathologic blood state or history of exhausting work and visceral disease, rather rapid course, absence of luetic history, combination of spasticity and ataxia, absence of painful nerves, or tenderness.

The course is dependent upon the virulence of the causative agent. In severe pernicious anemias and leukemias the patient may live only a few weeks. In the chronic forms these patients may live for several years, but their low vitality renders them easy victims of any accidental infection. A few cases have been reported cured, where the symptoms appeared rapidly during anemia, and subsided upon the disappearance of the latter. It is questionable, however, whether they belong in this category.

The treatment consists in the early detection and eradication of the toxic factor if possible. The later stages should be treated as chronic myelitis in the manner described above. Oppenheim has recommended the X-ray to the spine in the leukemic states. We may attempt to modify the course of the disease by vigorous tonic treatment with quinin, arsenic, strychnin, and transfusion of artificial serum and normal salt solution. Unfortunately, these measures are not very successful.

POLIOMYELITIS, INFANTILE PARALYSIS

The term poliomyelitis means inflammation of the gray matter of the spinal cord. As such inflammation is almost invariably restricted to the anterior horns, it is quite unnecessary to prefix the word anterior to indicate the locality of the gray matter implicated in the disease known clinically as infantile paralysis and essential palsy of children. The name has taken a definite place in nosonomy, however, and therefore no studied attempt should be made to dislodge it. For a long time following the recognition of poliomyelitis, both clinically and anatomically, it was believed that the disease occurred exclusively in children. It is now universally recognized that poliomyelitis occurs during adult life, and in many books separate description is given of its occurrence at this age. This is quite unnecessary, however, for the causation, clinical course, outcome, and treatment of the disease are the same in both instances.

Poliomyelitis may be acute or subacute in its development and course. A chronic variety has been described as the equivalent of spinal progressive muscular atrophy. It is dependent largely upon the use put upon the word inflammation, whether or not the gradual shrinkage, atrophy, and decay of the ganglion cells in the anterior horns which form the anatomical basis of this variety of muscular atrophy shall be called an inflammation.

The Causes of Anterior Poliomyelitis.—Anterior poliomyelitis is an acute infectious disease which prevails sporadically, endemically, and epidemically, due to an organism which causes directly, or indirectly through its toxins, an exudative inflammation of the gray matter of the cord. Its epidemic occurrence is much more common than formerly. The infection attacks chiefly the central nervous system and the mesenteric lymph nodes, but it has been found in the mucous membrane of the nose and throat and the mucous secretions of the stomach and intestines. The common and principal lesion is restricted to a very narrow area of the gray matter, almost exclusively of one or both anterior horns. The lumbar enlargement is the favorite seat of the disease, but the cervical enlargement is frequently affected. Rarely does the focus of disease extend to more than two segments of the cord, but inflammatory foci may occur simultaneously at different levels, such, for instance, as in the lumbar enlargement and in the cervical enlargement. Many instances have been recorded in which the inflammatory process extended in the gray matter throughout the entire cord; indeed, any segment of the cerebrospinal axis may be affected.

The essential morbid process in this disease is an acute inflammation manifesting itself predominantly in the veins and lymph channels of the gray matter, and affecting the nearby ganglion cells and neuroglia. Histologically the inflammatory foci are composed of lymphocytes, occasional polynuclear leukocytes, transition forms, fibroblasts, and rarely plasma cells. The extent and destructiveness of the inflammation are related to the virulence of the virus and the resistance of the individual, and may be confined to a few segments or may extend as small foci throughout the length of the cord, involving the spinal roots and meninges as well. In fact, the cases with no early meningeal and posterior root affection are rare.

The inflammatory process has a decided tendency toward limitation, and the tissues in which it occurs have a remarkable inclination toward restitution. It is very probable that the resistance of the ganglion cells of the spinal cord is very much greater than is usually considered. The structural changes which the cells undergo from the combined effect of the infection and the interference which it causes in their nutrition cannot be described here; but they are essentially those of cytolysis. Cells that are completely destroyed never recover, which is in keeping with the fact that the tissues having a specific function are never regenerated. Many of the cells in an inflamed area, which for a considerable time are functionally derelict and completely incapacitated, recover after the inflammatory process has subsided, the lymph circulation is restored, and the inflammatory exudate is absorbed. It is highly improbable that post-inflammatory cicatricial tissue, if so it may be called, undergoes sufficient secondary change to cause material destruction of the ganglion cells which remain after the inflammation has disappeared. It is scarcely necessary to say that, when a ganglion cell of the anterior horns of the spinal cord perishes, all of its intraspinal and extraspinal parts go with it. Therefore, the anterior roots, the peripheral nerves which they go to form, and the intramuscular distribution of these nerves within the actively contractile part of the muscle—in other words, the entire peripheral motor neuron—decay when the cell body is destroyed by the inflammatory process which forms the anatomical basis of anterior poliomyelitis.

The cause of poliomyelitis has been fully demonstrated to be a minute virus, non-filterable, which cannot be strained by any known filter, and hence cannot be isolated. This virus can be recovered from the nasal and oral mucous membranes, and the spinal fluid of patients suffering from poliomyelitis, and the disease reproduced by means of its inoculation in monkeys. It enters the central nervous system by means of the venous and lymph channels leading thither, and it has also caused the clinical picture when injected into the sheaths of peripheral nerves. In man the portal of entry is most probably the nasopharyngeal mucous membrane, which also serves as the point of egress. It is transmitted by carriers who may contract the disease or be entirely free from it. Recently the stable fly (*Stomoxys calcitrans*) has been accused as the transmitter of the disease.

It frequently follows public thoroughfares, in the epidemic form, and also appears at different points along railroad lines.

It sometimes follows in the wake of the infectious diseases; it occurs during the late summer, and especially in seasons of extreme sultriness; fatigue, excitement, exposure to wet and cold, overheating, and in general factors that exhaust the nervous system are oftentimes immediate precedents of the occurrence of the disease, both in the infant and the adult. In one recorded instance the disease followed a wound upon the knee, to stanch the bleeding of which moss from the roadside had been used. The vast majority of cases occur between the first and fourth years.

The recent epidemics in this country and abroad have given added impetus to the study of the disease, clinically as well as etiologically, and a great many different forms have been described by Wickman, Peabody, Draper and Dochez, Levaditi and Landsteiner, Flexner, and a host of others. The reader is referred to these authors for a detailed description of the clinical types. Suffice it to say here that there are still differences of opinion as to the proper classification of types. Wickman, for instance, recognizes eight types: (1) spinal, (2) cases simulating Landry's paralysis, (3) bulbar or pontine forms, (4) encephalitic, (5) ataxic, (6) neuritic, (7) meningeal, (8) abortive forms. As a matter of fact, the four types of the disease that we encounter may be spoken of as the abortive, the spinal, the bulbo-spinal, and the cerebral.

Symptoms and Course of the Disease.—The premonitory symptoms of the disease are those common to all the milder forms of infection, such as apathy, disinclination to play or work, somnolency, feeling of stupidity, and a desire to be left alone. Within a few hours, a day or two at the longest, the patient has considerable rise of temperature, reaching 102° or 103° F., which lasts from twenty-four to forty-eight hours in children and oftentimes longer in adults. It may or may not have been preceded by chilly sensations, but it is usually accompanied in children by manifestations of irritation of the central sensory sphere, such as vomiting, convulsions, flightiness, irritability, opisthotonos, strabismus, and a serious disinclination to be handled, as if severe pain were caused thereby. After these symptoms have lasted from one to three days, it is noted that the patient is paralyzed in one or all of the extremities, usually one of the lower extremities. As time goes on, what seems to have been loss of function of an entire extremity may disappear and the paralysis restricts itself to a single muscle or a group of muscles, especially those functionally associated. At this time atrophy of the muscles begins to show itself with some distinctness, although for many days the limb may retain its normal contour, and particularly if there is much subcutaneous fat. But the fact that it is atrophying is plainly apparent to the touch. Before and during this time the functions of the bladder and rectum have been disordered. This, of course, will be evident only in patients who are of an age to be observant of the calls of nature. In the beginning of the disease the patient feels sore all over and there is invariably complaint of great pain. But after the general phenomena of inflammation subside, there are no sensory disturbances, subjective or objective, although enduring pains in the extremities have occasionally been noted. When such symptoms occur they indicate an involvement by the inflammatory process of the posterior cornua and the root fibers passing therein.

As the individual muscle or group of muscles continues to atrophy certain accompaniments easily recognized on examination develop. These are loss of the tendon jerks, such as the knee reflex, when the muscles through which these reflexes are manifest are diseased, and some degree of reaction of degeneration to the faradic and galvanic currents, both in the nerve and in the muscle. The degree of completeness of the degenerative reaction varies in every case. When the atrophy is profound, there is usually complete absence of contraction both to the faradic and to the galvanic currents, but in other instances some faradic excitability of the muscle remains, a possession that augurs well for the recovery of the part. The atrophy usually reaches its height in from four to eight weeks after the occurrence of the disease; but it becomes more striking after this because in greater contrast to the progressively growing limb of the other side or groups of muscles of the same extremity. If the atrophy has involved an entire limb, no considerable deformity may result save that of a withered, undeveloped extremity; but usually the atrophy, limiting itself to a group of muscles, is followed by very marked deformity, particularly of the feet, different forms and degrees of clubfoot, which are caused by the unopposed contraction, the basis of muscle tonus, in antagonistic groups of muscles. The prevention of such deformities forms an important part of the treatment of poliomyelitis.

The prognosis is unfavorable if complete recovery is the standard by which such forecast is made. As a rule, the life of the patient is not endangered unless the inflammatory process is of great extent and profound severity, when the respiratory muscles may be involved. Practically all of the so-called abortive forms of the disease terminate in recovery. In about 20 per cent. of the spinal cases complete restitution follows. The prognosis, as a matter of fact, varies with the severity of the infection and the extent and seat of the pathological process. When the brunt of the disease is borne by the upper cervical cord and oblongata the prognosis is extremely bad.

TREATMENT

The treatment of poliomyelitis may be considered under four headings: 1. The prophylactic treatment. 2. The treatment of the febrile period. 3. The treatment of the immediate manifestations of the disease, particularly the muscular atrophy and its consequent paralysis. 4. The treatment of the remote consequences of the disease, deformities.

Prophylaxis.—The recent demonstration of the contagious nature of the disease has taught us that prophylactic measures are of the most vital importance. We should adopt as rigid precautions against the spread of the disease as possible, and should always personally insure their fulfillment. Poliomyelitis has already been placed on the list of diseases that must be reported to local health authorities in many states in this country and abroad, and the time is not far distant when this will become universal. The patients should be isolated and the other children in the family should be kept from school at least eight weeks. The rooms should be fumigated with formalin candles, and all discharges from the nose, throat, bladder, and rectum should be sterilized by heat and burning whenever possible. Those not affected should have their nasal and oral mucous membranes sprayed with 5 per cent. hydrogen peroxid or a dusting powder recommended by Levaditi and Landsteiner: Menthol, gr. 0.2, salol, 5 gr., acid boric, 20 gr. (menthol, 3 gr., salol, 65 gr., boric acid, ℥v) insufflated two or three times a day. Urotropin, gr. v—gm. 0.30—t. i. d. has been recommended for the unaffected members of the family. There is little unanimity of opinion concerning the time quarantine should be enforced. The Paris Academy of Medicine recently recommended the isolation of convalescents and patients for three months. It is supposed also that the isolation of one family, except the bread winner, for three months prevented an epidemic in Nebraska in 1909. Flexner has shown that the period intervening between inoculation of monkeys and the appearance of paralytic symptoms may be from two to three days to five weeks. In view of this, it seems to us advisable to adopt isolation measures of six weeks for all members of a family that are not bread winners. The chief difficulty in arriving at a satisfactory position in enforcement of prophylactic measures lies in the fact that not merely the patients are contagion carriers, but also healthy, unaffected individuals who have come in contact with the disease.

Treatment at the Time of the Attack.—The appropriate treatment during the febrile period is an ice-bag over the spine, or, if this is impossible on account of the age of the patient, the application of dry cups or a few leeches; the exhibition of repeated small doses of calomel, followed by a saline; and the administration of salol and phenacetin to control the fever, relieve pain, and to combat intestinal fermentation, and urotropin in large doses because of its possible entrance into the cerebrospinal fluid. Unfortunately, however, in the majority of instances, no suspicion is had of the nature of the disease, unless it be prevailing epidemically, until the febrile period has passed and the paralytic manifestations call attention to the fact that it is a spinal cord affection. The occurrence of convulsions calls for inhalations of chloroform, the use of a lukewarm bath, and perhaps the administration of a few doses of one of the bromin salts. As the pain and tenderness are often very great, only the most expert and gentle handling should be employed. The terror that is often manifested by these little sufferers is not due, as has been formerly suggested, to cerebral irritability, but actual pain of inflamed nerve roots, and this should always be borne in mind. Plaster of paris jackets and plaster beds have been suggested to make them more comfortable, but this method has not created a favorable impression as yet. When the tenderness is not too great to permit handling the child, great relief will be obtained from prolonged warm baths or packs, 30-45 minutes at 98° F.

After the fever has terminated, the child begins to feel well with surprising promptness and manifests an inclination to play and a desire to get up. Unfortunately, the mistake is oftentimes made of allowing it to do so. The patient should be kept in bed for at least a fortnight after the fever has disappeared, for during this time disintegration and absorption of the products of inflammation are going on in the cord. The completeness of this absorption has a bearing upon the degree of recovery. During this time the avenues of elimination from the body should be mildly stimulated, the patient's digestion and assimilation carefully attended to, and medicines given that are known to have the property of facilitating absorption. Ergot and its derivatives were recommended by the older writers, and faithfully copied by many of their successors, but its administration is more apt to do harm than good. During this stage, when the inflammatory focus can be accurately localized in the cord, mild counterirritation over the spinal column at a level corresponding to the seat of the disease may be used both in the child and in the adult, always bearing in mind that infants are very sensitive to such procedure.

Treatment of the Early Consequences of the Disease.—After from two to four weeks, treatment directed toward counteracting the atrophy of the muscles and toward improving the nutrition of the paralyzed parts should be undertaken. The most valuable agencies for this purpose are electricity, massage, local warmth, and exercise. Electricity is used in poliomyelitis with two objects: First, and least important, to influence the circulation of the blood and lymph through the focus of disease and thus to contribute to its recovery; and, second, to stimulate the peripheral neuromuscular apparatus to prevent inactivity atrophy and to increase the nutrition and volume of the fibers in the atrophic muscle that are not destroyed. The first indication is met by the use of galvanism, the positive pole being applied over the spinal column at the level of the inflammatory focus, the negative pole to the soles of the feet, the chest, thighs, or some other indifferent area, and a current of about five milliampères allowed to pass for five minutes twice a day. The usefulness of this procedure is not comparable with the application of electricity to the atrophied parts which constitutes the second indication. Either faradic or galvanic electricity may be used. The choice of current is determined by the degree of degenerative reaction. If the muscle and nerve are totally unresponsive to the galvanic current, very little benefit is to be expected from its use, although the stimulation of the cutaneous blood vessels of the part which it causes may be provocative of some benefit. The faradic current should be used in cases in which there is some response to it. Usually there are some fibers in a paralyzed muscle that respond to this current, and they, as well as the muscles of the immediate environment which are of similar function, should be sought out and stimulated to contraction. Two-inch electrodes should be used and kept in movement over the affected muscles, while a current of from two to four milliampères flows. As absorption of the inflammatory exudate goes on in the diseased focus, more and more muscular fibers will be found to respond to this current and probably also to the galvanic current, and these should be carefully encouraged and coaxed. The most serviceable way of utilizing the faradic current is by means of the faradic bath. This should be given at least once a day, preferably in the evening, as a part of the daily hygiene of the child's life. Followed by rubbing and friction of the affected part it has a beneficial effect not only upon the atrophied limb, but upon the entire nutrition. Electrical treatment should be persevered in for a long time, even though but slight improvement is manifest. Several cases have been recorded in which the persistent, continuous use of electricity (given, of course, in connection with other measures looking toward the improvement of local and general nutrition) has brought about surprising degrees of recovery, even after the atrophy had existed for a long time. One word of warning must, nevertheless, be given concerning its application. When the child is greatly frightened and is extremely rebellious to its use, it should be completely discarded until the little one has been educated to take it gracefully. The violent emotion and profound struggling which attempts at its use cause in many children are harmful, and more than counterbalance the benefit that the most sanguine can expect from its application. If the trouble be taken to put the electrodes on the body just as if the electricity was really being given for a number of times, until the child recognizes that such procedure is not attended by pain or discomfort, and then a very mild current allowed to pass, it will not be very long before a sufficient voltage of electricity may be used without objection on the part of the patient. As a rule, the strength of the current should be regulated by the feelings of the patient and the apparent effect that it produces.

Light massage may be instituted soon after the febrile period is passed. It should consist of mild kneading and pinching of the atrophied parts, followed by friction and percussion to influence the circulation. Its utilization should be accompanied by gymnastics given according to the Swedish method, the object being to give the diseased muscle, as well as the muscles that are functional antagonists of it, exercise and so prevent the one from undergoing changes the result of inactivity, and the other the result of unopposed action; in brief, the Swedish movements are thus passive and active, the patient resists the movements caused by the operator, and at the same time indulges in voluntary movements which the operator resists. Massage and resistance exercise should, like electricity, be kept up for à prolonged time, and they may be utilized simultaneously, or courses of the one and then of the other taken in succession. In large cities it is usually possible and always advisable to send the patient to an institution or gymnasium where these measures are scientifically and methodically carried out.

An important adjuvant to thwart the progress of the atrophy in a member is local heat. The surface temperature of the paralyzed extremity is usually from one to two degrees lower than in the unaffected, and the limb is cold, objectively and subjectively. The patient should always wear flannel underclothing and stockings, and at night should have hot water bags, hot water bottles, a soapstone, or some other substance that retains heat for a long time, in apposition to the withered extremity. During the day it should be plunged in hot water, once or twice, and oftener if convenient. Other means of adding to the local temperature and stimulating the circulation will suggest themselves to every physician to meet the indications in a given case.

The fact that the patient's limbs are incapacitated prevents him from play, getting about in the open air, and from taking exercise, which are so necessary for the maintenance of a high degree of bodily tone and nutrition. These must be replaced so far as possible by the utilization of baths and douches which have a tonic effect, attention to digestion and elimination, by an abundance of fresh air during the day and night, by regulation of time devoted to rest and sleep, and, in brief, by attention to the patient's general hygiene.

Prolonged warm baths at body temperature or 95° F. are also of material benefit at this stage. The effect is twofold: it keeps the entire body surface at an even temperature, warming the paralyzed limbs, and by the higher specific gravity supports them to a certain extent, and allows the patients to move them more freely than they otherwise would. The children should be encouraged to play and amuse themselves with toys while in the bath, and it is often surprising how much movement will be seen in apparently completely paralyzed limbs. Of course, this procedure should be carefully controlled, the water kept at an even temperature, and the patients should be watched for any signs of depletion. If they bear it well, it should last one-half to three-quarters of an hour and be repeated every day. It is best to follow it with gentle massage and friction.

Just how much effect strychnin has in counteracting the atrophy of the muscle cannot be estimated. There seems to be a consensus of opinion that it has some. It may be given internally with quite as much benefit as subcutaneously. Its administration should not be begun before all inflammatory phenomena have subsided. Its usefulness is probably dependent on its qualities to stimulate nutrition through the sympathetic nerves.

Treatment of the Remote Effects of the Disease.—The fourth indication, the treatment of the deformities that are so apt to follow the occurrence of poliomyelitis, requires the coöperation of the orthopedic surgeon, both after and during their occurrence. It may be taken for granted that, unless the atrophy is so extensive as to result in a uniformly withered extremity, deformity will be a remote consequence of the disease, and therefore the physician should be on guard to prevent it. The commoner manifestations of this deformity are some variety of clubfoot, particularly equinovarus. When the patient begins to walk around with the aid of crutches, the foot naturally drops, and, if the extensor muscles of the foot be paralyzed, the unopposed flexor muscles produce a deformity which, rather mobile in the beginning, is soon fixed and may require section of the tendo Achillis and forcible breaking up of adhesions before any orthopedic apparatus can be applied. If, however, the foot is fixed in a position of moderate hyperextension, by means of a gypsum bandage, an anterior splint, or a simple apparatus consisting of a collar above the ankle and another around the ball of the foot, connected on the anterior surface by means of a rubber band, or any contrivance fulfilling the same purpose, this may be obviated. The principle underlying the application of orthopedic apparatus in poliomyelitis after the patient has begun to get about is to prevent the deformity which results from the activity of muscles functionally opposed to those that are paralyzed. Later in the disease prosthetic apparatus having for its purpose to aid the patient in getting about or in the use of a limb must be applied. Many patients are helped to a life of great usefulness and are allowed to indulge in many pleasures which presuppose sound limbs, such as riding and bicycling, by these means. It is impossible to state specifically just the kind of prosthetic apparatus that will be required in every case. It depends entirely on the location and degree of atrophy and the nature of the deformity which results.

The importance of subjecting suitable cases to early tenotomy of the contractured muscles which cause the most incapacitating deformities can scarcely be overestimated. Division of the tendons of the contracted muscles is often followed by improvement in the nutrition of the paralyzed part. This is probably due to the increased movements of the parts which freedom from contracture and deformity permits. It is not necessary always to make complete or multiple tenotomies. Oftentimes partial section of a tendon is sufficient to allow of the adaptation of deformity-correcting apparatus.

The deformities of the spinal column that occur after an attack of poliomyelitis are those usually described as static or compensatory. If the patient has a short leg, when he walks there is a tendency for him to fall toward the side of the deficient member. To overcome this he at first consciously and afterward unconsciously leans toward the opposite side. This in time produces a lateral curvature. In an analogous manner the spinal deformity in the lumbar region known as saddle back develops when the muscles on the anterior surface of the thigh are affected. These deformities rarely require any orthopedic appliance if they are counteracted early by appropriate gymnastics. As a matter of fact, nowadays, when a department of gymnastics is an essential feature of every institution devoted to the treatment of the crippled, these deformities are not nearly so common among the poor as they were in former times. The utilization of gymnastic exercises requires a teacher of experience. There is no difficulty in finding such persons in every city, even though they are relatively few. If it be impossible to secure the services of such a one, some member of the family should make a pilgrimage to a teacher of gymnastics, and then utilize the knowledge acquired for the benefit of the patient.

Many surgical procedures have been suggested to overcome the condition known as flail joint, which occurs in the articulation of an extremity that has been withered by poliomyelitis. In some cases it may seem advisable to perform arthrodesis, that is, to cause artificial ankylosis, in order that the patient may have a firm basis of support. For instance, if the ankle joint is so devoid of its muscular and ligamentous support that the foot wobbles about in every direction, the artificial ankylosation of this joint may give the patient a useful extremity. Naturally, an operation of this sort should not be undertaken by the general practitioner, and therefore specific directions are not given here. It must suffice to call attention to the occasional necessity for its production.

Another surgical procedure that is sometimes used to much advantage is tendon grafting, or "function transference," which was proposed by Nicoladoni in 1882, and which has since been done quite extensively. The end in view is to attach or graft on to paralyzed muscles the tendon of a neighboring healthy muscle. The technique of the operation will vary, of course, with the muscles paralyzed. Therefore, each case requires separate consideration and ingenuity. If, for instance, the shoulder and upper arm muscles are involved in the atrophy of poliomyelitis, the patient may be entirely devoid of the power of flexion and supination of the forearm. In such cases it is proposed and has been accomplished to cut the tendon of the biceps and to implant it in the muscles of the forearm lower down. The *sine qua non* of its performance is that the muscle or group of muscles to which it is attached must be possessed of a fair amount of power in order that the requisite force may be obtained. The muscle that is functionally most nearly allied to the paralyzed one should be selected. Eve has shown that in some cases it is advisable that an antagonist of the paralyzed muscle be grafted with a view to weakening the action of the muscle whose unopposed contraction is causing the deformity. The method of uniting the tendons proposed by Goldthwaite seems to have found most favor. It consists in splitting longitudinally with a tenotomy knife the tendons of the paralyzed muscle, and drawing the divided tendon of the functioning muscle through the hole just made, union being effected by a few sutures passed through the tendon.

Determined use of these various measures will contribute to such restoration of function and so facilitate the unfortunate patient's recovery from this distressing disease that he may earn a livelihood and partake of many of the pleasures of his healthy brother.

THE PROGRESSIVE MUSCULAR ATROPHIES OF CENTRAL ORIGIN

Progressive muscular atrophy is the name given to a progressive wasting beginning in certain muscles or groups of muscles and extending to different parts of the body until it finally ends in death, either from marasmus or by the involvement of muscles whose activity is necessary for the performance of vital functions. The atrophy is dependent upon degenerative changes in the nerve cells of the ventral part of the cerebrospinal axis. The clinical form which the atrophy takes depends upon the groups of cells that are diseased, and upon the levels at which they are affected. The three most common seats of the disease are the cervical cord, the oblongata, and the pons. Gradual decay of the cells in each of these regions produces respectively the symptom-complexes characterizing: (1) Spinal progressive muscular atrophy (the so-called Aran-Duchenne type of the disease); (2) progressive bulbar paralysis, or glossolabiolaryngeal paralysis; and (3) progressive ophthalmoplegia, or gradual wasting of the musculature of the motor oculi nerve. These three varieties of progressive muscular atrophy are attended by the occurrence of individual symptoms which give rise to three distinct clinical pictures. In treatises on neurology they are considered as three distinct diseases. So far as they are known, the pathogenesis, causation, clinical accompaniments, course, and termination are the same in each one; and for this reason we consider them together. They are neuronic diseases in the strictest sense of the word, that is, the morbid condition underlying them is a progressive, gradual decay of the peripheral motor neurons, starting in the cell body or in the dendrites of the cells in the ventral part of the cerebrospinal axis, and extending throughout the neuraxons or axis cylinders to the actively contractile part of the muscular substance in which the neuraxons terminate. In other words, the spinal progressive muscular atrophies are chronic degenerative diseases of the peripheral motor neurons, with primary involvement of the cell bodies. There may be as many clinical types as there are groups of cells or functionally associated groups of cells in the cerebrospinal axis. Although the ventral cells of the cervical enlargement, the oblongata and the pons, are most frequently the seat of this degenerative disease, either singly or collectively, not infrequently, compared with the occurrence of any one of them, the cells of the lumbar or lumbosacral cord are diseased, giving rise to a clinical type of the atrophy which corresponds to the cells involved.

When the morbid process affects simultaneously the cell bodies of the peripheral motor neurons in the ventral horn and the terminal arborization of the central motor neurons (in other words, the endings of the neuraxons that come down through the cord as pyramidal tracts), the degeneration extends centrifugally in the peripheral motor neurons, and centrifugally and centripetally in the central motor neurons, to constitute anatomically a destruction of the cells of the anterior horn and a degeneration of the pyramidal tracts. This process may develop in any part of the spinal cord, but the location that it has special affinity for is the cervical region. The process usually extends from here into the oblongata and occasionally in the pons and farther toward the origin of the pyramidal projection. This morbid condition gives rise to progressive muscular atrophy, that has a distribution entirely parallel to other forms of progressive muscular atrophy dependent upon lesion of similar cells in the ventral horn, but in addition and owing to involvement of the pyramidal tracts (which involvement is not sufficiently severe to cause complete impairment of their conductivity) it gives rise to the phenomena of spasticity. The disease, therefore, which this lesion causes is a progressive muscular atrophy plus spasticity of variable degree. Because of the fact that it depends upon lesion of the anterior trophic gray matter of the cord and the lateral conducting white matter of the cord, it is known as amyotrophic lateral sclerosis. Without entering into any discussion as to whether or not amyotrophic lateral sclerosis is entitled to separate nosological consideration, we discuss its treatment under the general heading of progressive muscular atrophies, but at the same time state what is known concerning its etiology apart from these conditions.

The progressive muscular atrophies occur under two very different auspices: (1) An acquired form; and (2) a family form. The truth of this statement has but recently dawned upon the profession. Formerly it was believed that the progressive muscular atrophies were acquired or accidental diseases. Then an hereditary form of spinal progressive muscular atrophy was described; a familial form of bulbar paralysis and of ophthalmoplegia, and finally a familial form of spinal progressive muscular atrophy. Gradually, however, it has become apparent that the columns of motor cells in the ventral portion of the cerebrospinal axis may be so defectively developed, or immaturely constituted—the result of heredity—that they die in certain levels at variable times after the birth of the individual, varying from the first month up to the age of late maturity. When the cells of the lumbar enlargement die in early infancy and in more than one member of the family, we call the disease a family type of spinal progressive muscular atrophy, and the same when the cells of the cervical enlargement are diseased. Under similar circumstances, when the cells of the oblongata disappear, we call it the family type of progressive bulbar paralysis; and, when the cells of the ocular nerves atrophy, under similar circumstances, we speak of the clinical manifestations as a family form of ophthalmoplegia. In a treatise of this kind it is unnecessary to speak of all the clinical varieties of the progressive muscular atrophies in detail, so we shall discuss only the more important etiological features and the treatment of the different clinical types.

ACQUIRED SPINAL PROGRESSIVE MUSCULAR ATROPHY

(*Type Aran-Duchenne*)

Before the recognition of syringomyelia, localized hematomyelia, and chronic poliomyelitis, and before the time that intraspinal tumors were differentiated, it was believed that this variety of progressive muscular atrophy was much more common than it is now known to be. That it is the rarest of spinal cord diseases is conceded by all. Few, if any, have shown a willingness to follow the lead of Marie, who states that the disease has in reality no existence; for, after all of the conditions that are capable of giving rise to a similar symptom-complex are excluded, there still remains a small number of cases in which the diagnosis of progressive muscular atrophy due to destruction of certain groups of cells in the ventral spinal cord must be made. The symptoms that attend the development of such decay in these cells will depend upon the groups of cells involved and upon the severity of the morbid process. It has previously been said that the disease is primarily located in the majority of cases in the lower cervical region. This causes an atrophy of the muscles of the hand, principally of the interossei, the thenar and hypothenar eminences, which allows the hand to assume gradually a typical claw-like appearance. The atrophy extends to involve the muscles of the forearm, the shoulder girdle and arm, and, later still, the musculature innervated by the motor cells of the oblongata. The atrophy may finally involve the trunk and lower extremities, pointing to the implication of corresponding cornual cells. The muscular atrophy is attended by fibrillary twitchings, which are severe in proportion to the severity of the trophic process. The unopposed muscles pass into a state of more or less contracture, depending upon the rapidity of the atrophy in the affected part, and there is functional inability of a part or an extremity proportionate to the degree and extent of the contracture. If the trophic process is a rapid one, there is true reaction of degeneration to the faradic and galvanic currents in the neuromuscular apparatus; but, if it is slow and insinuating, as it is usually, the electrical reactions are quantitatively diminished, or there is only partial reaction of degeneration. There are no other symptoms save those attributable to and dependent upon the depreciation of vitality and nutrition which is coexistent with the disease. These, however, are oftentimes pronounced and are the only ones that are at all amenable to treatment.

The course of the disease is progressive, but not uniformly so. It develops in an insinuating way, and continues by irregular exacerbations until it renders the parts absolutely functionless. Then the force of the disease seems to be spent, but after a variable time evidences of involvement of contiguous or remote groups of cells appear, and the symptoms thus induced continue until the respective part becomes useless. All this time the vitality is getting gradually impaired, until finally, through a continuance of this, or through the appearance of the disease in muscles the functioning of which is necessary that vital processes may go on, the patient succumbs.

Etiology.—The causes of this variety of the disease are unknown. It occurs more often in men than in women, and especially during the years of mature adult life. It is rarely seen in any other classes than laborers and artisans. It has been attributed to recent and remote injury, both of the parts that show the atrophy and of the spinal cord; but it is unlikely that trauma has any determining influence, nor has exposure to cold. On the other hand, the relationship of occupation to the occurrence of the disease is a well-established one, and theoretically we can assume that there is an exhaustion of the peripheral neurons supplying the musculature that is put to excessive activity in smiths, polishers, embroiderers, etc. The occurrence of the disease has likewise relationship to the infectious fevers and to some of the metallic poisons, especially lead.

Syphilis until recently has been considered of no etiologic importance in the disease, but the Wassermann reaction has been found positive in a number of clinically pure types by ourselves and Spiller. Dana also found a history of syphilis in 33 per cent. of his cases. It remains to be determined whether these are instances of true syphilitic affection of the anterior horns or the result of syphilitic disease of the anterior spinal artery. Suffice it to say that in every case both the blood and the spinal fluid should be carefully studied for evidences of syphilis.

Treatment.—The treatment of acquired spinal muscular atrophy is a forlorn chapter in therapeutics. There is an impression abroad that the disease can be brought to a standstill by the use of strychnin given hypodermically in large doses. To a certain extent our own experience corroborates this view. We have had under observation for sixteen years a patient in whom the atrophy seems to have come and remained at a standstill after such treatment, combined with the use of faradic electricity, massage, and general hygienic measures. We have treated two other patients in the same way with encouraging results. But it has failed in several other cases. The suggestion to use strychnin emanated from Gowers, and he is more enthusiastic in its recommendation than almost any other writer. The nitrate is the preferable salt to use, and it should be given in from one-eightieth to one-sixtieth of a grain, and gradually increased until the dose is brought up to one-eighth of a grain, depending upon the results which attend its administration, and continued for a period of from two to four months. If symptoms of improvement do not follow such a trial it should be discarded, except as it may be used to meet certain symptomatic indications. Apart from this nothing has been recommended that approaches specific medication. The most important measures in the treatment are rest of the muscles that are beginning to atrophy, the use of electricity and massage to prevent the superimposition of inactivity atrophy, and the maintenance of a high degree of nutrition by regulation of the diet, exercise, hygiene, rest, and sleep, and the general state of the patient's bodily and mental health. So far as the causal therapy is concerned, it goes without saying that there should be at once a cessation of the occupation under the auspices of which the disease developed, and, if any relationship can be traced between infectious diseases or intoxications and the occurrence of the disease, medicinal measures should be taken to counteract and overcome them. The uselessness of electricity and massage in the treatment of progressive muscular atrophy is unswervingly contended for by some, but it is the experience of most physicians and our own that, when used with moderation, they are agencies of considerable value in delaying the progress of the disease. Massage is more serviceable than electricity. In using massage only the gentlest kneading movements should be employed. A very weak faradic current should be applied daily to the affected muscles for about five minutes. The danger is that too strong a current will be used. If the faradic current does not cause any response, the galvanic current should be used, both to redden the skin and to cause very slight contraction in the muscles. The real danger from the use of electricity lies in the exhaustion produced in the already severely affected muscles, and, unless it can be most skillfully applied, its use should be avoided entirely. To maintain the general nutrition use must be made of mild forms of tonic hydrotherapy, of exercise, of tonifying medicines, such as arsenic, iron, small doses of mercury, and iodid of potassium.

Cacodylate of sodium in doses of ¾-3 grains—gm. 0.016-0.195—hypodermically every day for twenty doses has been warmly recommended. Whatever effect it has is purely tonic in character, since it exerts no direct influence upon the course of the disease. It is best administered daily for twenty doses, then withheld for a similar period. Given in alternation with strychnin nitrate, it often has a remarkably tonic effect and is well worthy of consideration in every case.

The animal extracts have been recommended, particularly extract of the thyroid gland, but the published experience seems to be decidedly against it. Change of climate, the visiting of various health resorts, and sea voyages, all of which are not infrequently advised, are useless, save as they may contribute to the maintenance of the patient's morale and courage. Unlike its opposite, tabes, or progressive degeneration of the peripheral sensory neuron, whose progress is almost invariably hindered by persistent, indefatigable treatment, progressive muscular atrophy is not infrequently accelerated by anything approaching active treatment. It remains to be said, therefore, that the results of treatment should be carefully watched, and, if the disease is progressing, all attempts at treatment directed specifically to the atrophic process should be interdicted, while the therapeutic efforts are centered in maintaining the general health.

The family type of spinal progressive muscular atrophy is reverted to here merely for the sake of completeness. So far no therapeutic measure has been suggested that has the slightest effect in staying or modifying the course of the disease. Clinically there are two types of this affection, one that corresponds in the distribution of the muscular atrophy with the Aran-Duchenne type, and, secondly, the more frequent variety, in which the muscular atrophy begins, apparently, in the muscles of the lumboabdominal girdle, and successively involves those of the hips, the thighs, and the legs. The disease is characterized by its beginning in infancy or early childhood; by its manifestation in more than one member of the same family; by its progressive course; by the symmetrical location of the atrophy; and by the absence of any other symptoms than those due to the decay of the peripheral motor neuron. The disease ends fatally in from one to three years.

Chronic Progressive Bulbar Paralysis

(*Labioglossolaryngeal Paralysis*)

Clinically this disease consists, as its name implies, of a paralysis of the lips, tongue, and larynx, causing a destruction of some or all of the functions of these parts, associated with atrophy, particularly of the lips and tongue. Anatomically it is dependent upon a progressive atrophy of the motor nuclei in the ventral portion of the oblongata. The clinical phenomena of the disease are gradual disturbance of articulation, characterized by slowness and indistinctness; difficulty of mastication and of swallowing; in brief, difficulty in executing any of the movements subserved by the musculature supplied by the ninth, tenth, eleventh, and twelfth nerves. The inability to close the mouth and to pucker the lips gives to the lower half of the face a characteristic expression, while the atrophy of the lips and tongue, accompanied by fibrillary contractions, is apparent to the eye and to the touch. As the disease progresses, the manifestations of labial and lingual prehension, articulation, mastication, swallowing, and laryngeal activity become more and more impaired, while evidences of encroachment upon the lateral nucleus of the pneumogastric are manifest by attacks of cardiac palpitation and syncope. There are no sensory disturbances. The electrical excitability of the muscles is the same as in the spinal form of progressive muscular atrophy. The actual causes of the disease are unknown. Like progressive spinal muscular atrophy, the disease occurs in individuals who have put the musculature supplied by the peripheral motor neurons of the oblongata to exhaustive use, and the degenerative changes in these neurons are the natural successors of exhaustion. Thus the disease has been observed in glass-blowers, buglers, and cornet-players. Progressive bulbar paralysis is a rare disease at any age, and particularly so in the young, except the familial form, which will be referred to later. Occasionally it is seen in advanced life. The disease occurs about one-third more frequently in males than in females, and the cases observed in females develop at relatively a more advanced age. Factors which are often held responsible as causative of degeneration in other parts of the nervous system, such as rheumatism, syphilis, and gout, cannot be claimed as etiological factors in this disease, it being rare to find that the poison of these diseases has ever found a foothold in the system; nor can it be said that the disease is closely associated with degeneration of blood vessels, aside from the fact that it commonly occurs at an epoch when arteriosclerosis usually takes place.

The exciting causes are, first and most important, overexertion, particularly of the mouth and vocal apparatus, fright and anxiety, enervating habits, exposure to cold, and all forms of depraving influences. Theoretically, it is considered that toxic factors may be operative in some cases, but the only proof of such that can be advanced is one of analogy. In a few cases, however, it has been observed that the disease occurred after lead poisoning, diphtheria, and influenza. But in considering these cases it must be kept in mind that many of them were reported at a time when the symptom-complex now described under bulbar neuritis was unknown. Occasionally degenerative bulbar palsy seems to develop secondarily to acute inflammatory bulbar paralysis, just as progressive muscular atrophy seems now and then to follow many years after a poliomyelitis of infancy. Not infrequently progressive bulbar palsy is merely an extension upward of the degenerative process that is at the bottom of spinal progressive muscular atrophy and amyotrophic lateral sclerosis. A gliomatosis of the central canal extending into the fourth ventricle and the development of a tumor in the oblongata may likewise cause the syndrome of bulbar palsy. Very rarely the formation of an islet of multiple sclerosis or a number of them in the ventral portion of the oblongata may cause the syndrome of this disease.

The duration of the disease is a very variable one. Some cases run a uniformly progressive course, and terminate fatally within one or two years. In other cases the course of the disease is characterized by periods of improvement, or at least by remission of some of the distressing symptoms. Such remissions are temporary, and do not influence the eventual fatal outcome, although they may add to the patient's days and comfort. Very rarely, probably never, does the progress of the disease come to a standstill. The course is essentially chronic, and month after month the gradual increase in the intensity of the symptoms, notwithstanding the most assiduous treatment, is lamentable and discouraging. It is uncommon for the disease to take more than from three to four years to run its course, but occasionally it lasts more than twice that length of time. The immediate cause of death is universal exhaustion, death occurring from heart failure, attacks of syncope, or the pneumonia of swallowing; foreign substances, principally those taken for alimentation, pass into the larynx and into the respiratory passages and cause strangulation and suffocation, bronchopneumonia and localized pulmonary gangrene.

Treatment.—Although this disease leads uniformly to a termination which no therapy is able to avert, and although oftentimes our most strenuous efforts to delay the fatal outcome are negative, nevertheless, in the majority of cases, not only can the patient's comfort be contributed to, but his existence materially prolonged by assiduous and proper treatment. The real causation of the disease being unknown, it is impossible to speak of causal or prophylactic treatment other than to say that occupation or injurious indulgences that may possibly have any influence upon the disease should be interdicted and avoided. The most important factors in the treatment of chronic progressive bulbar paralysis are the maintenance of the patient's nutrition and the securing of as nearly as possible complete rest to the muscles that are undergoing atrophy. A semisolid and liquid diet should be adopted from the beginning, and this of the most nourishing kind. Milk and its various preparations, eggs, raw or slightly boiled, the most concentrated meat soups and nourishing gruels, should form the principal part of the dietary. The amount of force required to masticate and swallow meat and the consequent exhaustion more than counterbalance any benefits to be derived from it. The proteids, although important energizing agents and tissue builders, are not so urgently required as to warrant giving them in the shape of meat that must be chewed and swallowed. Proteids that admit of being given in liquid or in semisolid form fulfill every requirement. Careful diet lists should be prepared and the form of food changed with sufficient frequency to prevent the patient from tiring of it. It is a mistake to consider that a larger amount of food than is necessary to keep up the patient's weight is of any considerable benefit. It is advisable to remove dry breadstuffs early from the dietary, as they are most liable to enter the glottis and provoke severe attacks of spasmodic coughing. Semisolids are swallowed with greater ease than liquids. As soon as swallowing becomes especially difficult, resort should be had to the feeding tube. The diminished sensibility of the palate and vault of the pharynx which these patients have during the later stages of the disease contributes to the ease and comfort with which the tube can be passed. It cannot be too strongly emphasized that this mode of feeding should not be left until the patient is absolutely incapable of making deglutitory efforts. This mode of feeding may often be supplemented by limited rectal alimentation. As a rule, all forms of alcoholic drinks are harmful in this disease. Their ingestion tends not alone to make the patient more uncomfortable by contributing to palpitation of the heart and flushings, but they have a depressing after-effect, which is very bad. Any beneficial influence they have to stimulate the nutrition is easily obtained from the administration of a mildly alcoholic or non-alcoholic malt extract. The same may be said of tea and coffee; cacao, however, is a nutriment of real value. The patient should be prevented from using his voice with the same scrupulousness as in pneumonia. The early formation of the habit of communicating desires and thoughts graphically can only be advantageous and it is to be commended. Feeble efforts to dislodge food that gets between the teeth and cheeks by the tongue, as well as all other unnecessary movements performed by the labioglossolaryngeal musculature, are to be deprecated.

The two therapeutic measures which can be made use of by the physician with the best prospects of affording some relief are electricity and strychnin. Various ways of applying the former have been advised. Any benefit to be derived from this procedure is obtained through its preservative influence on the degenerating muscles, and not in any way on the degenerative process in the oblongata. Therefore, passing the galvanic current from one mastoid process to another, or galvanization of the cervical vertebral column, is not advocated. The use of the constant current to cause slight contraction of the muscles of the face, tongue, lips, and pharyngeal muscles, and to cause artificial swallowing movements for a few minutes each day, is the electrical procedure that is advised. As in all such degenerations, the danger is that too much rather than too little electricity will be given. The electrical treatment should be kept up every day for two months, each séance lasting for from five to ten minutes, and then an estimate taken of its effect. Particular warning must be given against the use of the galvanic current in this disease without a rheostat and milliampèremeter, since the same rule applies to the already exhausted muscles in this instance as in the spinal form. The patient's comfort and well-being are frequently contributed to by a moderate amount of general faradization, and by the use of massage. We have never been able to convince ourselves that massage of the atrophying parts was of the slightest service, but general massage, if given with sufficient mildness, may exercise a tonifying effect on the nutrition. Although the beneficial effects of strychnin are never so apparent in this disease as they are occasionally in its analog, progressive muscular atrophy of spinal origin, yet it is the most satisfactory vascular and muscular tonic available in chronic progressive bulbar palsy. It should not be given hypodermically. In many cases it can be advantageously combined with small doses of morphin, especially when the patient complains of dyspnea. The morphin, given in from one-thirtieth to one-fifteenth of a grain twice a day, acts as a reliable cardiac stimulant, while it exercises a soothing effect upon the patient's mind. The latter effect is well manifest in the relief of the dyspnea, which is almost always partly psychical. The use of iodid of potassium, mercury, and the salicylates, with the idea of specific and alterative action, as has been recommended by some writers, is a fallacy. Unless a history of comparatively recent syphilis or rheumatism can be obtained, or unless the serological findings indicate their employment, such drugs are harmful. Nitrate of silver, phosphate of zinc, and ergot have been used extensively, but they cannot be recommended.

Aside from steadying the nutritive balance by restoratives and aids to digestion and guarding the patient against factors that produce excitement or depression, the treatment is symptomatic. The patient should lead a quiet, uneventful life, as free as possible from strife, worry, and anxiety. Exercise in the open air, in moderation, is essential, but care is to be taken that it is not carried to the point of fatigue. The utilization of an occasional course of mild cold water treatment for its tonifying effects and to keep up the patient's general nutrition is advisable. The symptoms that not infrequently require particular treatment are drooling, coughing, dyspnea, syncope, cardiac palpitation, and hysterical types. Drooling is not so common a symptom as might be inferred from reading some of the older authors, but occasionally it is not only depressing and exhausting to the patient, but very annoying to those about him. It is but slightly influenced by belladonna and its alkaloid, or by any other medication save morphin. As it is not advisable to give the latter in quantities sufficient to affect the secretion, the drooling must be combated by absolute quiet of the patient. When, however, it seems to be very exhausting, no hesitation should be had in the use of morphin for a few doses. Attacks of spasmodic coughing and which are usually due to the entrance of foreign particles into the air passage, owing to incomplete closure of the glottis, are oftentimes a most annoying and exhausting symptom. It can be relieved temporarily by the administration of medicines that tend to blunt the sensation of the larynx, such as the bromids and morphin. But there is some danger in using these substances. The spasmodic cough is nature's signal that foreign substances are attempting to enter the respiratory passages. If the sensibility of the laryngeal mucous membrane is blunted, the entrance of such foreign substances may be unsignaled, and lead up to the occurrence of "swallowing" pneumonia. Despite this, small doses of morphin or cocain must oftentimes be used to combat the symptoms, but during their administration extra caution must be had in the feeding of the patient.

Dyspnea, syncope, and cardiac palpitation are all in the beginning of the disease partly psychical, and more may be accomplished for their amelioration by suggestion and by assurance that these symptoms are of no significance than by the administration of drugs. However, the effects of a cold water compress or ice-bag over the heart, the administration of a pungent aromatic cardiac stimulant, such as ammonia or ether, may be partly psychical as well as physical, and beneficial for both reasons. Hysterical attacks superimposed upon bulbar palsy are most distressing to witness, and extremely exhausting to experience. The treatment that is applicable to them does not differ from that which is serviceable in hysterical attacks occurring without organic disease. It has not seemed to me that the hysterical attacks have added to the gravity of the disease in two patients with chronic bulbar progressive paralysis, who have been for a number of years under observation.

If paralysis of the vocal cords or the entrance of foreign substances into the respiratory passages makes suffocation imminent, one should not hesitate to perform tracheotomy.

Family Form of Chronic Progressive Bulbar Paralysis.—The familial or hereditary variety of chronic progressive bulbar paralysis has been recognized only within recent times. It is apparently very infrequent, even compared with the variety just described. It occurs under practically the same conditions as the infantile and familial forms of spinal progressive muscular atrophy. It occurs in infancy and during the developmental years of life, and has no particular symptomatic features, aside from the ordinary form, save a participation in the atrophy and paralysis of the upper facial musculature. This is especially true of the cases of familial bulbar paralysis detected in infancy. Familial bulbar paralysis in the adult would seem to be unattended with involvement of the upper facial, but it has the unusual complication of muscular atrophy, especially of the muscles of the neck. As in chronic degenerative progressive bulbar palsy, the disease is a progressive one toward a fatal ending, but the course of the disease is oftentimes very slow, from ten to twenty years elapsing before the termination. The course of the disease is apparently uninfluenced by treatment, save in general and symptomatic indications pointed out for the idiopathic form. These should be followed out as consistently as possible in this form. The infantile familial variety is not infrequently superimposed upon the spinal variety of progressive muscular atrophy, or a forerunner of the former, and the treatment for the one is likewise the treatment for the other.

Asthenic Bulbar Paralysis

Asthenic bulbar paralysis, myasthenia gravis pseudoparalytica, bulbar paralysis without anatomical foundation, are the designations given to a class of cases in which the symptoms in their entirety resemble very closely chronic degenerative bulbar palsy, and in which after death—a termination to which the majority lead after a variable time—examination of the motor neurons as well as of other systems of the body fails to reveal any striking departures from normal.

Nothing is known of the causation of the disease. Of the cases reported the majority have been under the age of thirty, although it does appear during middle age and even later. It has been observed in a patient with profound chlorosis. The possibility that it is dependent upon a chronic intoxication of endogenous, or possibly of exogenous, origin has been suggested. There is much in the irregular course of the disease as it manifests itself in some patients that lends color to the view that it is dependent upon injurious agencies, the source of which is within the body. Various agencies have been regarded as etiologic factors. In many cases an enlarged thymus has been found, in others diseased parathyroids, in one case adenoma of the hypophysis. As the disease frequently exhibits symptoms referable to disturbed thyroid secretion, a relationship between the two has been sought. Disturbances of calcium metabolism, ammonia excretion, and creatinin excretion have been frequently observed.

The symptoms usually develop slowly. The patient may have complained for an indefinite time of easily induced fatigue and a feeling of overpowering exhaustion after comparatively slight effort. The development of the symptoms may, however, be rapid, so that the disease reaches its height in a few weeks. Frequently the initial symptom that attracts the patient's attention is ptosis of one or both sides. The ptosis may appear first on one side, then disappear, and the upper lid of the opposite side becomes affected, or it may occur on both sides simultaneously and be associated with paresis of some muscles supplied by the oculomotor nerve, such as the internal or superior rectus. Following this, or going before, there occur weakness of the muscles of mastication, paresis of the lower part of the face, and defect in articulation and in vocalization, which is associated with paresis of the abductors and adductors of the vocal cords. The voice becomes nasal and talking tires the patient and quickly exhausts his capacity in this direction. The lips are unwieldy and there may or may not be paresis of the tongue; swallowing is difficult or impossible, fluids regurgitate, and the soft palate is lax and responds very sluggishly to mechanical irritation. General weakness with a feeling of exhaustion in the trunk and extremities, true amyasthenia of all the motor parts of the body develops symmetrically, at the same time with or after the bulbar symptoms. In exceptional cases the weakness manifests itself first in the arms, extends to the legs, and eventually shows itself in the cranial nerves. As the disease progresses, and this it may do with considerable rapidity, respiratory and cardiac symptoms become very distressing and foreshadow dissolution. The course of the disease is irregular, made up of periods of remission and improvement and of periods in which the functions of the motor and sympathetic nervous systems are profoundly impaired. The manifestations through the sympathetic nervous system are practically those of shock and they are irregularly periodic in occurrence.

In contrast to true bulbar palsy the muscles preserve their volume, at least there is no true degenerative atrophy. Electrical irritability of the neuromuscular apparatus is preserved, but frequently exhausted after brief excitation, and irritability is not regained until after prolonged rest. There are no fibrillary twitchings of the muscles of the face and extremities, and the deep reflexes are present, but, like the electrical irritability of the muscles, their excitability is quickly exhausted and recovered only after rest. This is known as the myasthenic reaction and is an important aid in distinguishing the disease from others that simulate it, such as the bulbar form of disseminated sclerosis and polioencephalitis superior. There are no disturbances of sensibility, either objective or subjective, and the special senses are unaffected, although all of them become speedily fatigued. Digestion is impaired and normal intestinal activity is handicapped by lack of muscular tone. There is no drooling;

the sphincters are intact; and the psychical faculties are unimpaired. The shortest duration of any recorded case is six months. We had under almost daily observation a typical case for upward of eleven years.

Treatment.—Complete and absolute rest to all parts of the body, the eyes, the tongue, the throat, and the extremities, is the most important factor. Restoratives and the careful and judicious use of measures to maintain a high state of nutrition, while at the same time every precaution is taken to prevent unnecessary expenditure of energy and bodily waste, will be followed by the best results. Artificial feeding by means of the stomach tube should not be resorted to, as the movements of regurgitation caused by the passage of the tube are more exhausting to the patient than is the act of swallowing artificially masticated and liquid food. Oppenheim warns against the use of electricity for the purpose of causing muscular contraction, but recommends central galvanization. The usefulness of the latter has been corroborated by Goldflam, who reports the recovery of four patients. In the treatment of one case which has been very successful central galvanization was employed and the galvanic current to produce the weakest possible contraction of the muscles manifesting the asthenia.

If it is borne in mind that in this disease all the voluntary muscles, and especially the oblongata musculature, are in such a state that slight stimulation soon exhausts them, it will not be necessary to warn against the incautious use of strychnin, massage, and electricity, the three most available muscle tonics. All of these may be employed if intelligence directs their use. Strychnin should be given in extremely small doses, while its effects are carefully watched, and the moment it produces any feelings comparable to fatigue and exhaustion after its physiological effects have worn off the dose should be materially diminished. It is most useful when symptoms of inefficiency of the sympathetic nervous system are conspicuous. Another drug used to good advantage under like auspices is the salicylate of physostigmin in from 1-100 to 1-40-grain doses. Massage and galvanic electricity may, I believe, be used to advantage if care and attention are given to their application, and if they are given in sufficiently small dosage.

There is no specific diet to be recommended. Excess of carbohydrates, calcium in large quantities, and albumin have been tried, but with little success. Recently considerable attention has been paid to the ductless glands. Administration of suprarenal substance, spermin, thyroidin, hypophysis, and ovarian substances have all been employed with no relief, and, on the contrary, the condition has often been made much worse. Extirpation of the thymus when persisting has been recommended, but never tried. X-ray exposures over the thymus have also been of no avail.

Chronic Progressive Ophthalmoplegia

When the ganglion cells in the ventral portion of the pons undergo disease changes similar to those constituting the pathology of progressive muscular atrophy and progressive bulbar paralysis, the result clinically is bilateral atrophy and palsy of the external muscles of the eye, to which the name chronic progressive ophthalmoplegia is given. Like the other forms of progressive muscular atrophy, there are nosologically two varieties—the hereditary infantile form and the acquired idiopathic form. The acquired variety is the more common. The pathological change underlying it is similar to that of chronic bulbar paralysis and chronic progressive muscular atrophy, and it may complicate or be complicated by either of these two conditions, particularly the former. The variety of bulbar paralysis that is associated with chronic progressive ophthalmoplegia is, however, not so typical clinically as the uncomplicated variety. The same is true for progressive muscular atrophy of the Aran-Duchenne type. In other words, when the brunt of the lesion is borne by the cells of the pons and the oblongata, or the pons and the cervical cord, the resulting degeneration is neither so severe nor so extensive as it is when the pathological changes are confined exclusively to one of these segments. It would almost seem that the cause of the pathogenetic process when distributed over a larger area was insufficient to produce destruction of all the cells in a given area.

Chronic progressive ophthalmoplegia occurs under about the same auspices as the other two varieties of progressive muscular atrophy that have been described. It is most liable to develop in persons from twenty to forty years of age, and somewhat more often in males than in females. Intoxications and infections, as well as traumatism and exposure to cold, have been held responsible in some instances, but their relationship to this disease is no closer than is their relationship to the other progressive muscular atrophies. This, in truth, is very insignificant. It is more than likely that some of the cases that have been reported as occurring after diphtheria and poisoning by the minerals, such as lead, were dependent upon a rudimentary form of neuritis of the oculomotor nerve. It occasionally develops in syphilitic individuals who are benefited by the administration of antisyphilitic remedies, and thus this infection is considered of some causal importance. It is most commonly a complication or integral part of some other disease, such as general paresis, locomotor ataxia, multiple sclerosis, and the progressive muscular atrophies already mentioned.

The disease is evidenced clinically by the gradual and progressive occurrence of functional disability of the external muscles of the eyes which progresses until these muscles are completely powerless, usually associated with a slight or moderate degree of ptosis. The initial symptom is diplopia, but the patient soon unconsciously suppresses one of the images and depends upon monocular vision, so that after the disease has lasted for a time the patient does not complain of seeing double. The internal eye muscles are usually spared, but they may be involved to a considerable degree. The diagnosis can readily be made by process of exclusion.

The congenital variety is invariably associated with lack of development and functional incapacity of the facial nerve. This form of the disease is akin to the familial varieties of bulbar and spinal atrophy, that are supposed to be dependent upon incomplete development or atrophy of the respective nuclei. The course of the disease in this variety is apt to be more stationary and is sometimes associated with evidences of hypoplasia of other parts of the central nervous system.

Treatment.—Less can be done in the treatment of chronic progressive ophthalmoplegia than in any form of progressive muscular atrophy, and for the simple reason that we are unable to apply the measures, such as electricity, massage, and rest, that have some capacity to delay the progress of the atrophy in other varieties of progressive muscular atrophy. Unless there be some specific causation of the disease, such as syphilis or metallic intoxication, that allows of specific medication, efforts at treatment are limited to maintaining the general nutrition of the patient, and advising complete rest of the muscles involved. Strychnin does not seem to be of any use, except as a general tonic, nor do arsenic and iodid of potassium. When the disease occurs as a forerunner or concomitant of other diseases, such as have been mentioned, treatment must be directed toward opposing them.

Amyotrophic Lateral Sclerosis

The nosological relationship of amyotrophic lateral sclerosis to the progressive muscular atrophies has already been spoken of. This disease is characterized by the symptoms of progressive muscular atrophy of the Aran-Duchenne type, complicated with bulbar involvement, plus spastic paresis, particularly of the lower extremities, and exaggeration of the tendon jerks all over the body. The symptoms of spasticity usually precede those of atrophy, and it is therefore believed that implication of the terminations of the central or corticomotor neurons antedates that of the peripheral motor neurons. That this is so is shown not only by the occurrence of spastic symptoms before the trophic symptoms, but by the fact that, when cases come to autopsy, the morbid process in the central motor neurons gives every evidence of having been complete for some time, while that in the peripheral motor neurons is in progress. Although occasionally the disease is accompanied by pathological changes in other parts of the cord, such as degeneration of the posterior columns, as a rule the symptom-complex does not include any disturbances of sensibility or of the cutaneous reflexes, or disturbance of the functions of the bladder or bowels. The absence of such symptoms bespeaks the limitation of the disease process to the anterior horns and pyramidal tracts.

The causation of the disease is practically unknown. From analogy and from inference, particularly those based upon the findings in cases studied microscopically, it is believed that the anatomical basis of the disease is conditioned by some chronic intoxication acting through the vascular system. The forces that determine the involvement of the terminations of the central motor neurons and the beginnings of the peripheral motor neurons can only be conjectured. Such injurious influences as hereditary disposition of ganglion cells in different parts of the cerebrospinal axis to undergo decay without adequate cause, manifested by the occurrence of nuclear or neuronic diseases in the collateral ancestry; exhausting overwork of the extremities; trauma to one of the extremities or to the spine; vascular depravity following repeated exposure to cold, intoxications, and infections may have this effect, but we cannot prove it. As a matter of fact, infectious disease, syphilis, intoxications, cold, excesses, and the like have apparently very little relationship to the occurrence of the disease. It occurs in males and females with equal frequency, and develops ordinarily between the thirtieth and fortieth year. Occasionally the onset of the disease is rather abrupt and the patient succumbs in from twelve to eighteen months. As a rule, however, symptoms develop insidiously and the course of the disease averages from two to three years. It may, however, last much longer. It has been observed by Déjerine for 16 years, and by Flourod for 10 years.

There is a well-established familial variety of amyotrophic lateral sclerosis, which, like all other familial disease of this class, occurs in childhood and pursues a very chronic course, being oftentimes stationary for a number of years.

Treatment.—The treatment of amyotrophic lateral sclerosis is practically the same as that for chronic myelitis, in addition to the general measures that are of service in maintaining the nutrition spoken of under spinal progressive muscular atrophy. No drug medication has the slightest effect upon the course of the disease. The intensity of the spasticity may be somewhat decreased, and the suffering engendered by this condition mitigated by the use of prolonged lukewarm baths, in which the patient may remain for from two to four hours out of the twenty-four. The crippling of the patient through the spasticity and contractures that occur in the unopposed muscles after atrophy has become well pronounced can be overcome to some extent by the persistent use of active and passive gymnastics, but oftentimes the annoyance and fatigue attending such indulgence more than counterbalance the slight beneficial effect. Marburg very properly warns against division of the posterior roots for the relief of spasticity or contractures in these cases. When the morbid process invades the oblongata, the symptoms of bulbar paralysis should be treated in the same way as has already been mentioned under that caption. The same care must be expended upon the feeding, and all that this implies, that is necessary in true bulbar paralysis. The entire treatment of amyotrophic lateral sclerosis may be summed up in a word: make the patient as comfortable as possible. For patients who can afford it, this is most satisfactorily accomplished by providing them an intelligent nurse. Those who cannot should seek the shelter and care of a hospital. Despite this gloomy view of the treatment of amyotrophic lateral sclerosis, the physician should not despair. It is not too sanguine to expect that nature has provided a remedy to check the disease if it can be found and applied before the neural constituents, the decay of which forms the anatomical basis of the disease, have perished. This is surely true if the pathogeny of the disease is the result of some chronic intoxication. If the disease is a teratological defect, a disease of involution, it is idle to search for such a remedy.

MUSCULAR DYSTROPHY

The term muscular dystrophy is applied to that form of atrophy in which the primary changes are in the muscles themselves, in contradistinction to ordinary progressive muscular atrophy, in which the atrophy is secondary to disease of the ventral horn cells of the spinal cord, or the peripheral motor nerves. Formerly the progressive muscular atrophies and dystrophies were not differentiated, but so soon as it began to be recognized that the former were almost invariably acquired diseases of adult life, and that the latter were either family or hereditary diseases, occurring in infancy and early adult life, the distinction began to be made. In latter years the tendency has been to draw the line of separation very closely, from an anatomical standpoint, between the progressive muscular atrophies and the dystrophies. The result is a more or less widely disseminated belief that in the dystrophies there are no pathological changes in the spinal cord. As a matter of fact, it is highly probable that in every case of the latter affection there are secondary changes throughout the entire peripheral motor neurons, especially after the disease has existed a long time.

Anatomically the progressive muscular atrophies may be classified, according to the segment of the peripheral motor neuron that is affected, into myelopathies, neuropathies, and myopathies, according as the cell body, the neuraxon, or the intramuscular ending is primarily involved. In muscular dystrophy the lesion in the beginning is in the intramuscular nervous substance. The pathological changes that are found in the neuraxon and in the ventral horn cells are secondary and have nothing to do with causing the phenomena of the disease.

Formerly a number of clinical varieties of muscular dystrophy were described and much energy was devoted to the establishment of differentiations between the types; but to-day it is fully recognized that muscular dystrophy is a distinct disease subject to variations in the time of its development, in the groups of muscles which are affected, and, to a less important degree, in its clinical features. Separation of the clinical forms of the disease is of no service, save as it may facilitate prompt recognition of the disease and thus indirectly contribute to an estimation of the course and prognosis.

Many clinical types of progressive muscular dystrophy are described, but we shall refer only to the three important ones. These are: 1. Pseudohypertrophic paralysis, which occurs in early childhood, more often in males than in females, and which shows itself first in the lower extremities, especially the calves, by apparent hypertrophy of these and other groups of muscles, associated, however, with loss of strength. The enlargement of the muscles is due to a progressive lipomatosis which goes on at the expense of the muscular fibers and a myosclerosis. The disease then extends to the trunk, especially to the muscles of the sacrolumbar region, and gives rise to a characteristic gait, station, and mode of assuming the erect posture from a recumbent one. Later the upper extremities become affected, especially the muscles of the shoulder girdle and upper arm, and, as a rule, these muscles atrophy without undergoing a preliminary spurious hypertrophy. The muscles of the forearm and face are spared until very late in the course of the disease. As the disease progresses the apparently hypertrophic muscles shrink proportionately to the completeness of the fatty transformation and myosclerosis.

Leyden and Möbius individually called attention to a form of muscular atrophy that resembles this type in every respect save that the dystrophy is not attended with any considerable pseudohypertrophy. Some writers have endeavored to create a special type to which these clinicians' names are attached, but happily without success.

2. A facioscapulohumeral type, to which attention was first specifically directed by Landouzy and Déjerine. The atrophy, as the name indicates, reveals itself first in the face, especially in the orbicular muscles of the eye and mouth, which gives a peculiar cherubic expression to the countenance, known as the myopathic face and tapir mouth. It then extends to the shoulder girdle and arm muscles, very rarely to the lower extremities. This variety of dystrophy is not associated with any manifestations of pseudohypertrophy. It occurs in early childhood and occasionally at any time up to the period of adolescence.

3. Juvenile form. This variety has been particularly studied by Erb, and first reveals itself about the time of puberty, and has a distribu-

tion very similar to the facioscapulohumeral type, although the primary manifestation of atrophy is almost invariably in the shoulder girdle, while the face is involved later. Occasionally there is a slight degree of pseudohypertrophy accompanying the development of this form.

The accompanying table, taken from Sachs, shows the comparative similarity and differences of these three forms of the same disease:

TYPES OF PRIMARY DYSTROPHIES

	Muscular Pseudohypertrophy	Juvenile Form of Progressive Muscular Atrophy, Erb's Type	Type Landouzy-Déjerine
Part first affected.	Legs (calves).	Shoulder girdle.	Face and shoulder girdle.
Distribution of hypertrophy.	Calves, rarely thighs.	Muscles around shoulder girdle and pelvic girdle.	None.
Distribution of atrophy.	Thighs, deep muscles of back, shoulders and scapular muscles. Calves during later period; at that time also general atrophy.	Thighs, deep muscles of back, upper arm. Hypertrophied parts may become atrophic in later stage.	Face muscles, including lips and orbicularis palpebrarum; shoulder and scapular muscles.
Parts remaining normal.	Face, forearm, and hand.	Face, forearm, hand and leg muscles, except in last stages.	Forearm, hand, and legs, and deep muscles of back.

Treatment of the Dystrophies.—The inefficiency of therapeutic measures to cope with muscular dystrophy is in a large measure explainable by the fact that we are dealing with an evolutionary defect in the muscles, and, as there is very little borrowing from art to lend to nature, very little can be accomplished in delaying the progress of the disease after it has once become manifest, nor is there any way of preventing the disease except by the voluntary renunciation of procreation by those whose ancestral and collateral family histories show this disease. It should be kept in mind that not all of those whose ancestry or family reveals the existence of dystrophy develop the disease. Many of them do not. It is in such individuals that great care should be had that no strain or excess be put upon the neuromuscular system, which might awaken to reality the dormant dystrophic tendency. As muscular dystrophy in all its clinical varieties is a disease of infancy and early youth, it need not be said that these precautionary measures are to be taken particularly during the early years. It goes without saying that individuals who are afflicted with the disease should not marry. Those whose immediate family history shows the existence of the disease should be warned of the danger of reappearance of muscular dystrophy in their descendants, even though they themselves remain entirely free from it. Such individuals should be urged to remain unmarried, and, if they are married, to remain childless. If they insist on marriage, great care should be taken to point out their obligation to choose a mate that will, by means of healthy antecedents, tend to overcome the handicap and at least give a fair proportion of healthy children. As a matter of fact, the physician rarely has the opportunity of advising or applying prophylactic treatment except in those instances in which some member of the family is already under treatment for this disease. In such a case the apparently healthy children should be carefully instructed in the development of their muscular systems by systematized exercises. The difficulty is to choose a happy medium between overexercise, which might arouse the latent tendency to the disease, and insufficient exercise, which might allow the muscles to fall into a state of inactivity atrophy. The general care of such an individual should be directed to maintaining a supreme degree of nutrition.

After the dystrophies have begun to develop something can always be done, and often a very great deal can be accomplished in delaying the course of the disease by the proper utilization of gymnastics, massage, electricity, proper dietary and hygiene, and the coöperation of the orthopedic surgeon. All writers are seemingly in accord that systematic exercise is the most important measure. A few cases have been reported in which it would seem that the progress of the disease had been brought to a standstill by the use of this measure alone. It is impossible to say, except in a general way, what form of gymnastic indulgence will be beneficial in a given case. It depends somewhat upon the clinical type of the disease, upon the stage of the disease, and upon the age of the patient. As a rule, it may be said that some form of resistance exercise, carefully graded, particular care being taken not to put too great resistance upon the atrophied muscles or groups of muscles, is the most beneficial. It has the advantage that it may be employed in very young children who cannot be taught ordinary gymnastics. Although the cases that have been reported in which systematic active and passive exercise was followed by considerable benefit are of the facioscapulohumeral type and the juvenile type, theoretically there is no reason why it would not be as available in the pseudohypertrophic group. In these latter cases, unfortunately, the apparent hypertrophy is often taken by the family and by the physician to indicate excessive muscular development, and the disease is not recognized until it has passed into the moderately advanced stage of lipomatosis and myosclerosis. Then usually much time is wasted in desultory application of electricity before systematic exercise is begun. The truth is that up to date the most important measure in delaying the progress of muscular dystrophy is systematic exercise, and the sooner it is begun the greater is the prospect of improvement. The hazard attending its use is that it may be overdone. The physician should be content with comparatively slight results even after the expenditure of prolonged treatment.

Electricity is of very slight service in the treatment of the dystrophies. This may be explained in part by the fact that it is rarely applied as methodically and persistently as should be done to make legitimate estimate of its capacity to delay the atrophy. In muscular dystrophy the electrical reaction is quantitatively diminished in keeping with the degree of degeneration of the muscular fiber. There is no true reaction of degeneration. This, of course, allows the galvanic or faradic current to be used so as to produce muscular contraction, and such muscular contraction, if brought about carefully and without much intensity, is theoretically of service to prevent inactivity atrophy, and it may be of some use in delaying the specific atrophy. The greatest care must be exercised not to overdo the application of electricity. A few contractions in the atrophied muscles produced once or twice daily are far more advisable than a prolonged séance. The slowly interrupted faradic current is more advisable than the galvanic, especially in the early stages of the disease. The galvanic current has the advantage of stimulating the local circulation more than the faradic, and it may, therefore, be legitimately alternated with the former. The effects of electricity to improve the local nutrition are much more definitely obtained by the use of massage, which should be applied in the shape of very light muscle kneading to every case of muscular dystrophy. In the utilization of these three measures, exercise, electricity, and massage, sight should not be lost of the fact that in the interval of their application the patient should be as nearly as possible at complete rest. Nothing can be more injurious than the attempts of patients to drag themselves about when the lower extremities are the seat of muscular dystrophy, or to use the upper extremities in some occupation when the disease is of the juvenile type. This should not be construed to mean that the patient should not be in the open air, and under auspices that contribute to general tonification of the muscular system. The aid of the orthopedic surgeon should be sought just as soon as deformities arising from contracture of unopposed muscles interfere with the getting about of the patient. Such contractures should be overcome by partial or complete tenotomy and the parts retained in an approximately normal condition by the use of that prosthetic apparatus which is indicated. Winged scapulæ, if caused by serratus paralysis, may be anchored to the ribs by means of wire, etc., and a fairly useful arm obtained. Occasionally some such apparatus may be beneficial in giving support to the parts that are not deformed by the contracture.

During the past few years various animal extracts have been thoroughly tried in the treatment of dystrophy with the hope that benefit might accrue in some unknown way. Extract of the thymus gland has been extensively used, and, to lesser extent, that of the thyroid also. In our own experience there is not the slightest semblance of evidence that they are of any benefit.

In brief, the treatment of the muscular dystrophies consists in the employment of those physical measures that are known to tonify the muscular system; in the adoption of dietary and hygienic means that serve to maintain general nutrition, and the adoption of measures that overcome deformity and contribute to the comfort of the patient. So far no drug has been discovered that exercises any influence upon the course of the disease.

NEURAL PROGRESSIVE MUSCULAR DYSTROPHY

(*The Peroneal or Leg Type of Progressive Muscular Dystrophy*)

This variety of progressive muscular atrophy has been studied especially by Charcot, Marie, and Tooth, and is not infrequently referred to by the names of one or all of these investigators. During the past few years it has received extensive consideration based upon close clinical and anatomical studies, both here and abroad, so that at the present time the clinical delineation is well known and the morbid processes upon which it is dependent bid fair to be satisfactorily elucidated, although at the present time they are not entirely so. After the disease was first described it was contended by many writers that the morbid process was predominantly, if not exclusively, a degeneration of the nerves with secondary involvement of the spinal cord, particularly the columns of Goll; but at the present time there can be no doubt that the so-called "neural form" of progressive muscular atrophy is dependent upon different anatomical processes. In some cases it is primarily a disease of the peripheral nerves, while in other cases it is just as certainly a primary myopathy. Furthermore, it is wholly probable that in some cases the pathological changes occur simultaneously in the spinal cord and peripheral nerves, or even primarily in the cord. The clinical picture of this variety of the disease, it may therefore be said, does not depend upon an individual pathologicoanatomical foundation.

The symptoms of the disease are, it may be readily inferred, subject to variation in kind, in intensity, and in course. As a rule, the muscular wasting begins in the musculature of the peroneal nerves, and is manifested by the gradual occurrence of clubfoot. Occasionally the atrophy shows itself first in the upper extremity, especially the small muscles of the hand and the extensors and flexors of the forearm. Wherever the primary manifestations of the atrophy may be, it may extend to any part of the body. In a case recently reported by Siemerling, in which the initial muscular atrophy was of the thighs and the hands, there eventually developed loss of the pupillary reaction, nasal speech, tremulous voice, in addition to an extreme degree of paretic atrophy of the forearms and entire lower extremity. The distinguishing clinical features between this form of progressive muscular atrophy and the spinal form are the sensory disturbances which are never absent, and the more complete reaction of degeneration in the atrophied muscles to the electrical current. The causes of this disease, aside from the fact that it is a family affection, are entirely unknown. It usually begins during the early years of life, almost invariably before the age of puberty, and pursues an extremely chronic and irregular course. That there are exceptions to this rule is shown by the fact that Oppenheim and Cassirer have reported a patient in whom the disease began in the forty-second year, and Egger has described the disease as it occurred in two brothers, aged respectively thirty-three and thirty-eight years of age. The male sex is affected oftener than the female. The customary attributable exciting causes, such as exposure, intoxications by lead, alcohol, and syphilis, as well as the inherited diminished capacity of resistance of the nervous system, are spoken of in the etiology of the disease, but practically nothing is known of its real causation save that it is a family affair.

Treatment.—The treatment of this form of progressive muscular atrophy calls for the measures that have been enumerated in discussing the treatment of progressive muscular atrophy of spinal cord dependency and the progressive muscular dystrophies. The fact that all family nervous diseases pursue a much slower course, and are oftentimes characterized by more or less prolonged cessation of the apparent activity of the disease, affords opportunity for the use of electricity, massage, and gymnastics looking toward the retardation of the morbid process and the changes in the muscles. The fact that the disease usually begins in the feet and the legs, causing some variety of clubfoot which seriously cripples the patients, prevents them from getting the exercise and indulging in some of the pleasures of life that might otherwise be afforded. These deformities should be subjected to the same kind and grade of orthopedic treatment as similar deformities arising under other conditions.

MYOTONIA CONGENITA

(*Thomsen's Disease*)

The name myotonia congenita is given to a peculiar family disease first described by Thomsen, a Silesian physician in whose family more than twenty cases occurred. It is characterized by the occurrence of chronic contraction in all the voluntary muscles on attempt at innervation or movement, while at rest the neuromuscular system appears to be quite normal, save for the hypertrophy of the muscles which always exists after the disease has lasted for some time. The disease is classified as a family affection, but that it is not always familial has been proven by a number of recent reports. The hereditary factor in its causation may be manifest as a direct transfer from an ascendant or indirectly by inherited disposition. The predisposition to its occurrence may be atavistic. Jacoby[*], among others, has shown that the symptom-complex of the disease may occur independently of neuropathic heredity, developing after acute infectious diseases, such as typhoid fever and diphtheria, and transitorily after depraving influences, such as prolonged exposure to cold. He suggests that the names myotonia acquisita and myotonia transitoria be given respectively to these forms of the disease.

Myotonia congenita usually manifests itself in the early years of childhood, or at least before puberty, and frequently under the immediate auspices of fright, shock, or mental excitement. The essential feature of the disease is the occurrence of tonic spasm in the voluntary muscles on attempt at purposeful movements, and the inability of the patient to relax this tonic condition by force of the will. At the end of fifteen to thirty seconds the contraction relaxes spontaneously, and, after several repeated attempts at motion followed by a similar tonic spasm to a lesser degree, the patient is finally able to perform such purposeful movements, and for a long time, as walking, running, and dancing. The muscles present a characteristic mild tonic reaction constituted of normal mechanical, faradic, and galvanic irritability of the motor nerves, and increased irritability of the muscles. These, combined with absence of all symptoms pointing to a gross involvement of the nervous system, go to make up the essential feature of the disease. As in most neuropathic conditions, the occurrence of this disease is not infrequently associated with other symptoms pointing to an unstable nervous system, such as psychical symptoms, epilepsy, and migraine.

It is very doubtful that the disease can be looked upon as a congenital abnormality of the neuromuscular system, particularly in light of the fact that acquired and transitory forms occur. It would seem more legitimate to postulate an inherited or familial instability of this system which can be called into active morbidity by factors arising from within and without the individual. Such an instability of the neuromuscular system may also be acquired. In the congenital form very little can be done to prevent this instability, but much may be done to delay the advent of its manifestations. The patient whose birthright entails the potentiality of this disease should be advised concerning the selection of an occupation or profession, and concerning the questions of marriage and propagation. He should be warned against those indulgences and habits that have a tendency to increase the irritability of the neuromuscular system. Such are strains, exposure to excessive cold and heat, excesses in eating and drinking, mental excitement, and the like. It should be made known to such patients that a quiet, uneventful life may be full of usefulness to themselves and others, and the existence of their disease need not necessarily shorten their allotted days. No treatment has so far been suggested that is of any service in overcoming the manifestations of the disease, save the adoption of measures looking toward this kind of an existence. Naturally, electricity, massage, gymnastics, and Swedish movements have all been thoroughly tried. They do not seem to have any particular beneficial effect. Some physicians have claimed that they have noted amelioration of the disease from the use of massage and graduated gymnastic exercises, but the consensus of opinion is that they are of very slight service. In the acquired form causal therapy should be employed in addition to the general measures already mentioned.

SPASTIC SPINAL PARALYSIS

Both Erb and Charcot long ago described a clinical entity characterized by slowly progressive rigidity of the lower extremities, with exaggerated tendon jerks, and called it primary lateral sclerosis. It is extremely rare, so rare, in fact, that its very existence has been denied by many authors, but Erb's justification lies in the published reports of Strümpel, Déjerine and Sottas, and others. It occurs predominantly in males, appearing usually in the third decennium, although it has been seen at a much later period, and is so slow in its development that its progress is barely perceptible. The disease itself is not fatal; these patients always die from some intercurrent disease, and it may last until the senile period of life appears.

Primary lateral sclerosis is more nearly a monosymptomatic disease than any other in neurology, and is characterized by an insidiously progressive rigidity of the legs. These patients first realize that their gait is not so brisk and active as formerly. On arising after remaining seated for any length of time, they are stiff and perhaps a little awkward, which soon passes off after exercise. They become fatigued after long walks, and begin to experience difficulty in mounting or descending steps. From then on there is chronic intensification of this condition, until the gait becomes slow, with short, shuffling steps, pronounced adduction of the thighs, the toes catching in slight obstacles. Objectively there are marked resistance to passive movements, exaggerated tendon reflexes, patellar and ankle clonus, Babinski phenomenon, and Oppenheim reflex, i. e., extension of the great toe when the thumb or a blunt instrument is drawn from above downward along the inner surface of the calf with considerable pressure. The abdominal and cremaster reflexes are lively and never absent. There are no sensory disturbances, the bladder and rectum functionate normally, and there are no trophic disorders. Various forms of the disease have been described, but it is doubtful whether they really belong to the picture of true primary spastic paralysis. That the arms may be affected, and exhibit true muscular rigidity with increased tendon jerks, is probably the case. When one leg is more affected than the other, the corresponding arm is also more spastic (Oppenheim). Strümpel and others have described spastic bulbar symptoms, spasm of the larynx, and emotional disturbances, but we should always accept these forms with reserve, since we are more apt to be dealing with disseminated sclerosis or amyotrophic lateral sclerosis. This is also true of the various sensory disturbances described, except possibly the aching pains apt to accompany muscular rigidity.

The diagnosis is made from consideration of the slow progression, rigidity, and exaggerated reflexes. It may sometimes be confused with multiple sclerosis, when the latter begins insidiously with few symptoms, except rigidity and exaggerated tendon jerks. In the latter instance, however, the irregular progression of events, remissions, and absence of abdominal reflexes will serve to differentiate it from primary lateral sclerosis. It can be distinguished from beginning amyotrophic lateral sclerosis by the rapid progress and appearance of bulbar symptoms and atrophies in the latter. Before making the diagnosis of primary lateral sclerosis, one must be careful to exclude all residua of previous lesions, such as trauma, hemorrhages, transverse myelitis, cerebral infantile diplegia, etc. It is perfectly possible to find a low-grade spasticity persisting years after recovery from the above-named conditions, and a careful inquiry into the early life of these individuals will nearly always establish this fact.

The pathogenesis has never been demonstrated in a satisfactory manner; although the most reasonable hypothesis is that the myelinization of the pyramidal tracts, because of faulty development or congenital weakness, gives way earlier in the individual's life than the other systems. It is unable to withstand the demands of an ordinarily active life.

Pathology.—Our knowledge of the pathologic process in these cases has been contributed to by Erb, Strümpel, Déjerine, and Sottas and Spiller. The membranes are normal, the cord is usually of normal volume, and the cut surface shows normal color except in the lateral pyramidal tracts, where a grayish sclerosis becomes apparent. Histologically we find a simple descending degeneration in the lateral pyramidal tracts with low-grade neuroglia increase. The degeneration begins below the pyramidal decussations, sometimes in the cervical, sometimes in the dorsal, regions, and persists throughout the length of the tracts. In the absolutely pure cases the degeneration was limited to the lateral pyramidal tracts. In Déjerine and Sottas' case there were a mild degeneration in Goll's tracts in the lower dorsal portion and a pallor of the direct cerebellar tracts.

The vessels are normal, except in the sclerosed tracts, where they show a simple sclerosis.

Treatment.—We are as yet quite helpless in controlling or arresting the progress of this disease, and we must fall back on attempts to check its progress, regulate the life of the patient, and make him as comfortable as possible. Toxic doses of strychnin sulphate have been employed with no success. Iodid of potassium has no influence upon its course, and silver nitrate, gold chlorid, and the colloids have been of no avail. On the other hand, we should insist that these patients avoid fatiguing walks, should not be exposed to exhausting temperatures, cold or damp atmosphere. Alcohol and tobacco in moderation only and simple, wholesome food is the best diet. If the rigidity is very annoying and a tendency to contractures exists, prolonged—45 minutes to one hour—warm baths at 100° F. will give the most relief. Massage and passive exercise will maintain the nutrition of the spastic muscles, and give relief from the ache that so frequently accompanies this condition. Coarse vibration to the spine, setting up exercises, or severe manipulation may improve the circulation and afford temporary relief. It is always best to explain carefully to these patients the mechanism of their gait, and show them how in part it may be overcome by exaggerating every joint excursion. We have often seen as much improvement in the gait of these patients after practice in this as from any other measure.

SPINAL SYPHILIS

Spinal syphilis, lues cerebrospinalis, is for the most part so intimately associated with, or dependent upon, meningeal lesions that most writers prefer to treat it under the caption of diseases of the meninges. We prefer to treat it separately and at this time, because its many manifestations, its protean type, and its pathology entitle it to a place among the systemic affections of the cord. Although the disease is almost invariably dependent upon meningeal lesions, its clinical forms are often those of diffuse or simple systemic lesions in which the meningeal symptoms are of little or no significance.

Symptomatology.—The clinical appearances of spinal syphilis, of course, depend upon the character of the pathologic lesions causing them, and, as we have seen how manifold the latter may be, it is not difficult to conceive that the disease may simulate many of the well-known clinical types of neurology. They may be roughly grouped into forms caused by chronic meningitic lesions, where the symptoms will be more or less referable to root disorders. If the process is an active gummatous infiltration, the symptoms will be those of acute myelitis. Slowly infiltrating lesions starting in the leptomeninges cause system degenerations. Large gummata of the dura, non-infiltrating in type, cause pressure symptoms; while arterial disease may cause diffuse lesions in white and gray matter or isolated discrete patches, causing a clinical type which closely resembles multiple sclerosis. Then, finally, there is a diffuse infiltration of the leptomeninges which simulates tabes and frequently cannot be distinguished from the latter without serological examination.

The construction of a simple clinical entity would, in view of the foregoing, be quite a hopeless task; there are, however, a few symptoms that are constantly found in very many cases, and should always excite our suspicion when found. They are: Argyll-Robertson pupil, pain, bladder disturbances, and sexual impotence. They are of little value alone, but, when they appear together with other symptoms, their importance cannot be overlooked. In those forms of spinal syphilis in which the lesion is limited to the meninges and roots, pain is probably the most predominant feature. The pains may be severe, boring, dull, aching; or, if the posterior roots are pinched, the lightning, neuralgic form of pain will be the chief symptom. Girdle pain is not infrequent. The affections of the anterior roots may take the form of simple paralysis accompanied by stiffness, or there may be a complete flaccid paralysis with lost reflexes and reaction of degeneration. A favorite locality for the latter is at the level of the cervical enlargement, causing flaccid palsy and atrophy of one or more muscles of one shoulder girdle, or both. The paralysis may take the Déjerine-Klumpke form of Erb's shoulder-arm paralysis. Pain is always present at some stage of the development, although it is sometimes overlooked.

The symptoms of active gummatous infiltration depend upon the extent and locality of the lesion. Usually the clinical picture is that of incomplete myelitis, in which the Brown-Séquard syndrome and its variations are most prominent. On the other hand, the process may be confined to the posterior quadrants of the cord, and we find ataxic paraplegia with disturbance of all forms of sensibility. The reflexes may be present, abolished, or exaggerated. If the gray matter is invaded atrophy of the corresponding muscles will be found. The onset is usually rapid, often abrupt, and preceded by pain. The slowly infiltrating forms starting in the leptomeninges are most frequently classified under the title meningomyelitis, and they, as a rule, constitute the underlying pathological process of syphilitic spastic spinal paralysis. These may appear clinically as Erb's spinal paralysis, or there may be invasion of the other tracts, i. e., sensory tracts, and then ataxic and profound sensory disturbances become manifest. Oppenheim pointed out long ago that a modified Brown-Séquard syndrome may frequently be present in these types, and his statement has since been confirmed by many others.

In 1892 Erb separated a group of cases from the mixed forms and called them syphilitic spinal paralysis. This clinical syndrome is characterized by slowly progressive spastic paresis of the lower limbs, accompanied by pains, irregularly distributed areas of anesthesia or hypesthesia, bladder and rectal disturbances, usually in the form of difficulty in starting the stream, exaggerated knee and ankle jerks, Babinski and Oppenheim phenomena. At that time Erb expressed the belief that, although he could not base his claim upon histopathologic examinations, sooner or later the lesion would be found in the lateral pyramidal tracts and in the posterior columns as well, and that it would consist in simple primary degeneration of these areas. He reiterated this some years later and insisted that his contentions had been borne out by anatomic study. A number of other writers, principally Nonne, Henneberg, Minkowski, and others, have studied the situation with great care, and have come to the conclusion, best summed up by Nonne: there are in reality certain pure cases of primary syphilitic spastic paralysis; that the lesion is a primary systemic degeneration of the pyramidal tracts and posterior columns of post-syphilitic nature, and not due to focal degenerations in the columns.

Extradural gummata cause spinal symptoms by pressure, and they will vary in accordance with the direction and force of the pressure. Not infrequently very confusing clinical pictures are produced by a lateral oblique direction of the pressure.

Syphilis of the spinal vessels is capable of producing many varieties of symptoms. There may be sudden apoplectiform paralysis of the type caused by hematomyelia; there may be the typical symptom-complex of syringomyelia; there may be anterior horn lesions producing chronic progressive muscular atrophy, or a combination of the latter with system disease of the posterior columns or lateral tracts, thus combining the atrophies with spastic or ataxic phenomena, with bladder disturbances and hypesthesias or paresthesias.

Syphilitic disease of the blood vessels often causes small punctate foci of hemorrhage and degeneration in the tracts, and the picture of combined sclerosis results. Several cases of disseminated patches, luetic in nature, have been reported which clinically bore the marks of multiple sclerosis.

The tabetic form or pseudotabes, as it is called by some, so closely simulates tabes that it is often impossible to distinguish it from the latter. It begins shortly after the primary syphilitic infection and it develops rapidly; the symptoms of pain, girdle sensation, ataxia, Romberg, are very intense, and the progress is often startlingly hurried.

Diagnosis.—The diagnosis of spinal syphilis has been made much easier for us since the introduction of careful serological examinations. Definite types have been described by Plaut, Nonne, and Citron, and we are now fairly well able to distinguish this disease from non-syphilitic diseases of the central nervous system, as well as from tabes and taboparesis. Spinal lues differs from non-syphilitic diseases by the presence of lymphocytosis of the cerebrospinal fluid, marked excess of globulin, and positive Wassermann reaction in blood and fluid. The differentiation from tabes and taboparesis rests on the enormous number of lymphocytes—90 to 1,400 per c. mm.—and the tremendous increase in globulin in the former, whereas the percentage of positive Wassermann reactions is slightly higher. Whenever it is impossible to make these examinations, we are forced to rely on the rapid development, Argyll-Robertson pupils, bladder disturbances, and irregular distribution of the changes in sensation. The presence of cerebral symptoms is also of great help; the isolated root symptoms and irregular course enable us to distinguish the condition from chronic myelitis of other causation. It is to be distinguished from the non-syphilitic forms of combined sclerosis by the history of infection, presence of Argyll-Robertson pupils, optic neuritis, and other cerebral signs. The differentiation from multiple sclerosis is often impossible clinically, and can be made only by therapy.

Prognosis.—The prognosis depends upon several factors: first, the activity of the process; second, the location of the lesion and its character; third, its amenability to treatment.

(1) In rapidly infiltrating processes, where it has gone unrecognized for a long time, the prognosis as to both recovery and life is uncertain. We may by active antisyphilitic measures be able to check the specific disease, but unable to influence the progress of secondary degenerations. On the other hand, if recognized early enough, this process should yield promptly, and we should expect marked improvement and even recovery. (2) The location has great influence on the prognosis, for, if the lesion is within the gray matter or even in the meninges, repair of the damaged tissues is very slight, and we can only hope to arrest the progress. We may say that, wherever the lesion may be, if it is of the soft, infiltrating character it can be controlled by active medication and complete recovery may be expected. If it is of the slow chronic connective tissue type little or no impression is made on its course. This also holds true for Erb's type.

Treatment.—If the diagnosis of spinal lues is once established a vigorous antiluetic treatment should be instituted immediately. Even in cases in which the diagnosis is doubtful the same plan should be followed. The purpose of this is twofold: In the first place, we destroy or attempt to destroy the pathogenic organism and thus arrest the progress of the disease; and, in the second, we allow the system an opportunity to absorb toxic products, carry off deleterious matter, and repair the tissues that have been disturbed but not destroyed. There are three agents most useful for this purpose—salvarsan, mercury and its salts, and iodid of potassium. The specific action of the last named has often been questioned, and to it has been ascribed rather a rôle of alterative, assisting in repair and prevention of connective tissue formation. A true specific action upon Spirochæta pallida has recently been given it when used in large quantities by Neisser, but this we doubt and deny, at least as far as our experience goes. The selection of salvarsan or mercury as the antisyphilitic agency to be used rests with the individual preference of the physician. As said under another head, the use of salvarsan is still too recent to give final data concerning its position in our therapy. If employed, the first dose should be the full dose—0.6 gram—advised by Ehrlich, given intravenously, and should be repeated at intervals of one week to one month for four to six doses. If, because of optic neuritis, gastrointestinal disturbances, or marked debility, we prefer to give mercury, it should be given preferably as inunctions, gr. xx—ʒii—rubbed in thoroughly in the orthodox manner. Daily injections of 1-3, 1-4, or 1-6 grain of bichlorid or enesol, highly recommended by the French, may be injected daily, or salicylate of mercury suspended in liquid albolene, 1-2 grains once a week. Iodid of potassium, starting with grams 10, t. i. d., may be given in increasing doses up to the limit of tolerance of the individual. Minute, careful attention to the general hygiene and daily routine of these patients is very essential. Hot baths, while taking mercury, are very beneficial. They should be taken every week, as often as two or three times, and hot enough to induce perspiration. Whenever possible, these patients should be sent to one of the numerous baths—Arkansas, Virginia Hot Springs, or Richfield Springs in this country, Aix-la-Chapelle, Neuenahr, or Baden-Baden, in Europe—at the completion of a course of treatment. Often a mild course of tonic baths at home is beneficial.

For the relief of pains the same methods should be employed as in tabes dorsalis: Spinal irritation with cautery-blisters, faradic brush, or galvanism, massage, passive joint exercises, and prolonged hot baths for the spasticity.

If medical treatment fails to relieve pain, surgery must be employed. Nerve stretching, excision or evulsion of painful nerves, or section of the posterior roots must be tried.

Pathology.—The anatomic changes found in cerebrospinal lues are many and of widely different forms. The most frequent changes are found in the leptomeninges—diffuse infiltrating collections of small round cells which penetrate the cord *en masse* or as slender prolongations. In severe forms the piaarachnoid is swollen, infiltrated with round cells, forming a ring about the cord, compressing it and interfering with the circulation. The arachnoid alone may be invaded in the same way, the pia and dura remaining untouched. The inner surface of the dura may be the seat of these low-grade inflammatory changes, and they choose the exits of the spinal roots as point of predilection. Rarely are the entire cord and its coverings affected in this manner. It usually occurs at different levels and with varying degrees of intensity in the same individual. The vessels of the cord and membranes are for the most part the seat of active inflammatory changes, which consist in obliterating endarteritis, thickening of the media, hyalining degenerations, and perivascular lymphocytic infiltrations. The lesions within the substance of the cord are vascular in origin in the greater number of cases. They are not confined to any particular area, but may affect gray and white matter equally. Sometimes small punctate hemorrhages or extravasations may be the starting points of system degeneration, or intense glia reactions about the vessels and subsequent formation of small foci of softening with lymphocyte infiltration and granular cells. In the early stages the axis cylinders may show a true myelitic reaction, i. e., swelling of the medullary sheath, bursting of same into balls and buds of degenerated myelin, and loss of the axis cylinder proper. The gray matter may degenerate into small foci, which later become confluent and present the picture of syringomyelia. Occasionally true gummata of the dura are seen.

If the process is chronic, it may be confined to the dura and limit itself to slow progressive connective tissue proliferation, which later extends to the piaarachnoid, becomes hard, firm, and contracts. Histologically we find the dura metamorphosed in thick, fibrous connective tissue which is invading the piaarachnoid, poor in vessels, and shows a tendency to twist and form concentric knobs or warty growths. This is known as pachymeningitis hypertrophica.

Erb's type shows no true syphilitic changes. It is rather post-syphilitic in character, and consists in primary degenerations in the lateral pyramids, posterior columns, with slight degeneration in the periphery.

SYRINGOMYELIA

Syringomyelia is a disease of the spinal cord and oblongata, characterized clinically by an association of motor, sensory, and sympathetic symptoms, closely simulating tabes plus progressive muscular atrophy, and anatomically dependent upon cavity or fissure formation predominantly of the gray matter. The cavity or fissure of the cord may be single or multiple. It varies in diameter from a mere slit to an opening sufficiently large to admit the end of the little finger. Longitudinally it may extend throughout the greater part of the spinal cord, but it is more often confined to one or a few segments. The hole or slit or fissure or whatever it may be does not preserve the same shape throughout its entire course, nor does it occupy relatively the same position in different segments. The cervical cord is most commonly the seat of cavity formation, and after this the upper cervical region with the lower third of the oblongata. The lumbar segments are the next most common seat, while the dorsal segments are rarely involved—that is, by a cavity that confines itself to these segments, it being borne in mind that it is not at all uncommon to find holes in the dorsal cord in connection with cervical or lumbar cavities. When the syringomyelia is of the cervical segments the gray matter is usually rather uniformly encroached upon, while, when the cavity formation is of the lower dorsal and lumbar region, the posterior horns and posterior columns are oftener involved and there is relatively less encroachment upon the anterior gray matter. A very curious fact is that the anterior cornua never seem to be exclusively affected, nor are the anterolateral columns, although the corresponding parts of the posterior half of the spinal cord are frequently exclusively affected. The area of special predilection of cavity formation in the oblongata is the ascending root of the trigeminal nerve and the vagoglossopharyngeal-hypoglossal nucleus.

Syringomyelia is by no means a pathological entity. The cavity may be a congenital condition existing in the shape of an enlarged central canal. In some such instances the possessor goes through life without any apparent evidences of its existence. Such a condition must, however, be a locus of diminished resistance wherein inflammatory or degenerative changes may begin. The hole in the cord constituting syringomyelia may be due to a gliomatosis resulting in the formation of a glioma which has predominantly longitudinal extent, or it may be caused by a proliferation of glia tissue and consequent destruction of the parenchyma, that is, the condition known as gliosis. It would seem to be definitely proven that cavity formation may be the result of hemorrhage into the substance of the cord, which, acting by cleavage in the direction of least resistance, causes the formation of an empty space after the coagulum has been partially or completely absorbed. Syringomyelia has been found associated with chronic pachy- and leptomeningitis, with chronic myelitis, especially with the form known clinically as lateral sclerosis, and with other organic diseases. Just what relationship these morbid conditions have to the syringomyelia has not been determined. Occasionally it has been found coexistent with hydrocephalus, atrophy of the brain, cerebrum, and cerebellum, and with congenital conditions, such as spina bifida.

Very little is known of the etiology of the disease. Although of recent recognition, its occurrence is by no means very uncommon. Men are afflicted more often than women. A neuropathic history is the rule, and the disease has been encountered in several members of the same family. It is associated sometimes with such functional nervous diseases as exophthalmic goiter, hysteria, chorea, neurasthenia, and Raynaud's disease, but it is highly probable that these conditions are merely expressions of an encroachment by the cavity formation upon the sympathetic nervous system representation in the spinal cord. The most important attributed exciting factors are trauma and the infectious diseases. Just how these act, except to favor the occurrence of gliosis or gliomatosis and hemorrhage into the substance of the cord, it is impossible to say. Some authors have laid particular stress upon dystocia as an exciting cause. But it must be extremely uncommon, and when it has any influence is through producing rupture of intramedullary blood vessels. The infectious diseases, such as typhoid fever, pneumonia, and malaria, may likewise act to produce degenerative changes in the blood vessels, which predispose to intramedullary hemorrhage and thus to cavity formation. Syphilis plays no rôle in the etiology of the disease, although syringomyelia occasionally occurs in syphilitic patients. The endeavor has been made by some physicians to establish the nosological identity of syringomyelia and anesthetic leprosy, but very little success has attended such efforts.

Symptoms.—The typical symptom-complex of syringomyelia is progressive atrophy of individual muscles or groups of muscles, associated with a widespread partial sensory paralysis, manifesting itself as analgesia and thermoanesthesia, with fully preserved tactile sensibility, and with trophic manifestations, especially of the skin and of the bones. The seat of the atrophy will depend naturally upon the location of the cavity in the cord. Usually it is of the upper extremities and face. If the cavity is in the lumbar region, the atrophy will be of the lower extremity. The muscular atrophy is dependent upon a destruction of the ganglion cells of the peripheral motor neurons. When the cells constituting the common origin of the vagus, glossopharyngeal, and hypoglossal nerves in the oblongata are encroached upon, there will be muscular atrophy and other disorders indicative of the partial or complete destruction of these cells. The motor and sensory manifestations of the disease may be entirely or predominantly unilateral, or they may be bilateral. The dissociation of sensibility—that is, the occurrence of thermoanesthesia and analgesia with preservation of tactile sensibility and of the muscular sense—although not absolutely pathognomonic, as it may occur with tabes, hematomyelia, Pott's disease of the cervical region, hysteria, and divers forms of multiple neuritis, is by far the most unvarying symptom. If the lateral columns of the cord are encroached upon by the cavity formation, there will be rigidity and paresis of the extremities corresponding to the location of the cavities. The state of the deep reflexes will also depend upon whether or not this part of the cord is involved. If the group of cells from which spring the neuraxons supplying the muscles of the front of the thigh are encroached upon, the knee jerks will be absent. On the other hand, if they are not, and the lateral columns are affected, the knee jerks will be increased. In the atrophied muscles the electric contractility is diminished in proportion to the degree of atrophy, but true reaction of degeneration is exceptional. Oculopupillary manifestations, consisting of retraction of the eyeballs and narrowing of the palpebral fissure, and inequality of the pupils (Schultze eye of German writers), are very common and usually unilateral.

The trophic symptoms vary enormously in different cases. They consist of softness and pultaceousness of the skin, of such eruptions as erythema, eczema, and pemphigus; and of ulcerations, gangrene, and alteration in the nutrition of the nails. The cellular tissue may be the seat of phlegmon, abscess, and lacerations. The joints are sometimes the seats of indolent arthropathies, especially the shoulder, similar to those of tabes, and the spinal column is usually the seat of scoliosis or kyphoscoliosis. The more common vasomotor and secretory symptoms are dermographism, edema, cyanosis, and increase in the secretion of the salivary, lacrimal, and sweat glands. The sphincters and sexual functions remain intact.

A symptom-complex, known as Morvan's disease, is identical with syringomyelia. It consists of muscular atrophy and weakness of the upper extremities developing simultaneously with analgesia or anesthesia, extending over the arms, and associated with the occurrence of panaritium on the fingers, which leads to deep-seated ulceration and often to crumbling of the terminal phalanges.

The fact that anesthetic leprosy sometimes produces a syndrome very similar to that of syringomyelia has already been mentioned.

Treatment.—The treatment of syringomyelia consists essentially in protection from harmful environment, support of general body health by hygiene, hydrotherapy, and tonics, and by rigid control of and careful attention to the different deformities and trophic disturbances.

These patients should be warned against exhausting exercises, especially of the arms and neck; overexertion causing undue strain of the cardiac muscles. They should be carefully instructed concerning the sensibility disturbances and taught how to protect these parts against blows, extreme heat or cold. As hemorrhages into the cavities are apt to occur, they should be warned against sudden exertion, and also against joint strains, on account of the liability to arthropathies. It is well to have them always test hot water, hot bottles, etc., with normal parts before allowing the anesthetic areas to come in contact with these articles.

The general bodily health is best maintained by a simple, wholesome diet, out-of-door life, tonic baths, light exercise, and rest. CO_2 baths have been recommended for their stimulating effects. If there is much pain, caffein citrate, gr. i (gm. 0.065), antipyrin, gr. ii-v (gm. 0.13-0.30), dionin, gr. ¼-½ (gm. 0.016-0.032), pyramidon, gr. ii-vii (gm. 0.12-0.5), either singly or in combination, are helpful. Gentle passive stretching of the painful nerves has been advised. Mechanical apparatus should always be employed for this purpose. The milder cutaneous irritants, the violet ray for 10 minutes to the painful area, chloroform liniment, or vibration by one of the commercial machines, may prove efficacious. Suspension should never be employed, likewise lumbar puncture, unless absolutely necessary for the diagnosis, since the danger of hemorrhage is too great to warrant their employment. If there is severe spasticity, prolonged warm baths and passive movements are serviceable. Of the tonics, arsenic, strychnin, and ergot have received most attention. We have never seen actual benefit from the last-named drug. Arsenic, either in form of Fowler's solution, gtt. v-x, t. i. d., or arsenious acid, gr. 1-50 (gm. 0.002-0.065), t. i. d., or daily hypodermic injections of cacodylate of soda, gr. 3/4-1 (gm. 0.048), is of service in maintaining general tone. Strychnin sulphate has been often tried, but with uncertain results. One case was apparently arrested for at least eight years by the accidental administration of an almost lethal dose. The patient emerged from a night of convulsions with apparently no ill effects, and never as long as he was under observation displayed any evidences of progress. If given, it should be administered hypodermically, daily increase in the dose to the point of tolerance.

Ten-minute exposures of the spine to the X-ray have been recommended, but we should always bear the possibility of burns in mind. Radium baths have recently been recommended by Schlesinger, also 10 minutes' to one and one-half-hour exposure to radium rays.

The care of the trophic disturbances is mostly mechanical and surgical. Arthropathies should be protected by various orthopedic devices whenever support is needed; suppurations in the joints should be freely incised and attempts at ankylosis encouraged. Ulcers, perforating or otherwise, should be treated surgically, and care taken to prevent spread of the necrotic process. Operative measures are only to be undertaken after all other mechanical means have failed. We should always wait for spontaneous healing of ulcers and infected joints, since this is not at all uncommon. Bier's hyperemic method has been used successfully a number of times in the treatment of ulcers. Joints that are prone to subluxation should be protected wherever possible, and even arthrodesis may be necessary in the habitual cases.

MULTIPLE SCLEROSIS

Multiple or disseminated sclerosis appears in early adult life—in the second or third decennium—but may begin in early childhood, and has even been observed as late as the 45th year. The disease was formerly considered to be relatively infrequent, and, as regards the classic type described by Charcot, this is still true. Within the last ten years, however, numerous abortive forms (formes frustes of the French), atypical forms, and variations from the original type have been described, and the disease has taken a front rank in the order of frequency of neurological diseases. It is considered by most writers to be next in frequency to tabes dorsalis.

Etiology.—Unfortunately, we know little of the causative agents of the disease. It is very frequently seen after acute infectious diseases—typhoid, influenza, scarlet fever, measles, pertussis, cholera, acute articular rheumatism, and malaria. It has developed after pregnancy and metallic poisoning of lead, zinc, tin, and manganese workers. Trauma and severe colds also have a place among the etiologic causes. In the majority of cases, however, it is impossible to find a single cause, and we are forced to rely on Strümpel's theory of endogenous intoxication. Ziegler has spoken of an abnormal disposition to glia hyperplasia in these individuals.

Symptoms.—The classic type described by Charcot consists of stiffness, weakness of the legs, muscular rigidity, exaggeration of reflexes, Babinski and Oppenheim phenomena, hurried action of the sphincters, absent abdominal reflexes, tremor of the hands, usually intentional in character, ataxic movements of the hands, scanning speech, disturbances of vision, and nystagmus. In a great number of cases there are irregular patches in the optic nerves with contraction of visual fields and scotoma. The onset may be abrupt, with convulsive moments and unconsciousness, and these so-called apoplectiform attacks may occur during the disease. The mentality is in some cases severely affected, and may be confused with general paresis. There is very often emotional disturbance that may be taken for hysteria. All forms of sensory disturbances may be present, even the Brown-Séquard type of dissociation, but the characteristic feature of these disturbances in multiple sclerosis is that they are constantly changing from day to day in an almost kaleidoscopic fashion. Impulsive laughter is an infrequent symptom. Sometimes severe boring, sticking pains and paresthesias are complained of, but they never have quite the character of lancinating pains. Ataxia is very frequently present, and may be either cerebellar or spinal in character. The disease pursues an irregular course. It progresses for a time as if it were going to incapacitate the victim. Then there is a cessation of symptoms and he gets all but well and remains so for several months, perhaps even a year; then the activity of the disease reveals itself again and the patient soon becomes more helpless than he was before. In the majority of instances transient diplopia and subjective vertiginous states are the earliest symptoms. In every instance in which spasticity occurs in a young individual and no apparent cause can be found for it, multiple sclerosis should be thought of.

Treatment.—The treatment of multiple sclerosis presents many difficulties, and, as we know of no specific drug to arrest the development of the small patches, we are obliged to fall back on general hygienic measures, regulation of diet, etc. These patients should be warned against overexertion, mental strain, worry, and the like. The physician should so regulate their daily life that it is on an even plane. Long, exhausting work or exercise should be avoided; the bladder and rectum emptied regularly and at stated intervals. Rest in bed after a severe remission will often improve them greatly. Hot baths are to be avoided (Oppenheim), but tonic baths, salt rubs, and passive resistance exercises should be employed. These have the good effect of general supportive measures, improve the circulation in the spastic limbs, and help prevent contractures.

Among the drugs, silver nitrate (gr. ¼-¾, t. i. d.) and Credé's ointment have been extensively used. The latter is especially recommended by Oppenheim. Mercury has been employed, but should be used with great care, because of the tendency to optic nerve atrophy. Strychnin should not be used in most cases, because it tends to increase the spasticity. We have seen very good results follow the daily hypodermic administration of cacodylate of soda, ¾-1 grain. It appears to hasten remissions and unquestionably prolongs them. Veronal has been recommended for the tremor, but its constant use is a dangerous practice. As yet electricity has proved of little avail. Mild central galvanism may be lightly applied to the neck and spine. Opotherapy offers little hope as yet, and the same may be said of sera inoculations and vaccines.

The severe contractures are best treated by mechanical apparatus, or, if these are not successful, tenotomies and even section of the posterior roots may be necessary.

CHAPTER II

DISEASES OF THE PERIPHERAL NERVES

Howell T. Pershing

TREATMENT OF NEURITIS IN GENERAL

The word neuritis, meaning inflammation of a nerve, is here used to denote the morbid processes excited in nerve fibers by any injurious influence, whether physical, infectious, or toxic. Thus used it includes mechanical damage, sometimes amounting to complete section, and also processes which are degenerative rather than inflammatory, as in the neuritis of diphtheria or lead poisoning. There are certain lines of treatment applicable to all cases of injury or inflammation of the peripheral nerves, and in order to avoid needless repetition these will be considered first. The special modifications or additions needed for individual nerves will be given later.

Removal of Cause.—The first and most important indication for treatment is to remove the cause of the disease. In mononeuritis, or localized neuritis, which involves a single nerve or a few adjacent nerve trunks, local causes such as injury or localized infection predominate. In multiple neuritis, or polyneuritis, which involves many nerves in different parts of the body, only a correspondingly widespread cause can be sufficient, namely, a general infection or intoxication. Both general and local causes may act in combination.

In traumatic cases the wound is to be cleaned immediately and as far as possible rendered aseptic. If the nerve has been divided the ends should be found, by dissection if necessary, freshened, and brought together by fine sutures (Chipault, 6). This must be done at the earliest possible moment, otherwise the difficulties of the operation will be increased by the retraction of the severed ends and the changes in the wound, while rapid degeneration in the peripheral portion of the nerve will lessen the chances of ultimate success. Nevertheless, secondary suture even as late as a year or two after division may be successful. In case the loss of substance is too great to admit of the ends being brought together the gap may be bridged for a centimeter or two by catgut sutures, with a hope that the regenerating fibers will follow them (suture à distance). In rare cases where, on account of the proximal part of the severed nerve being inaccessible, or the gap being too great for suture à distance, it is impracticable to unite the peripheral part to its own stump, it may be stitched into a slit made in the sheath of a closely adjacent nerve, or a less important adjacent nerve may be cut, so that its proximal end may be united to the peripheral part of the nerve originally injured. The part of the nerve sutured should be enveloped if possible in a sheath of fascia to protect it from scar tissue and prevent adhesions. After suture of one of the nerves of a limb such a posture as will prevent tension is to be maintained by a suitable dressing for about three weeks. Pressure must of course be carefully avoided.

If the injury is caused by fracture of a bone its ends are to be brought into correct apposition and sutured, then the nerve is to be separated as far as possible from the fracture by the interposition of fascia or muscle. This must be done immediately, as the nerve degenerates very rapidly under continued pressure.

In case the nerve is injured, but not severed, and recovery seems to be prevented by the contraction of cicatricial tissue or the pressure of exudate, it may be necessary to perform the operation called neurolysis, that is, to expose the nerve and free it from adhesions, at the same time trying to place it in such a position or so envelop it with fascia as to prevent future adhesions.

Pressure, stretching, or irritation due to adjacent disease or new growth must as far as possible be removed by treatment of the primary disease. If infection has extended from a suppurating wound to a nerve, free drainage of pus is the first essential, after which every effort should be made to increase the systemic resistance to infection by means of fresh air, food, cheering mental influences, tonics, and an appropriate vaccine.

Constitutional causes are of the greatest importance in multiple neuritis, but they may also be of fundamental importance in neuritis limited to a single nerve. Syphilis, tuberculosis, and gout are especially apt to cause a localized rather than a multiple neuritis. Other important conditions most often causing multiple neuritis are alcoholism, diabetes, severe anemia, and poisoning by lead, arsenic, mercury, ptomains, etc. The so-called rheumatic or uric acid diathesis is often an important aggravating cause even when the neuritis is mainly due to other agencies. It specifically calls for the administration of sodium salicylate, aspirin, salophen, or some other form of salicylate. In any of these constitutional conditions, aside from the treatment of the specific infection or intoxication, which is described in its appropriate place in this work, free elimination is to be secured by means of laxatives, diuretics, and diaphoretics, having due regard, of course, to the patient's powers of resistance. In all forms of neuritis there is a marked tendency to nervous and constitutional debility which must be combated by a diet as nutritious, abundant, and varied as the digestive organs can be made to tolerate. If any restriction is necessary let it be in the carbohydrates. Proteins and fats are most essential.

Rest.—Next in importance to the removal of cause is rest of the affected area from every mode of activity, whether motor, reflex, or sensory. The more severe and more recent the disease or injury the more nearly absolute is the indication for rest. Only when the acute symptoms have begun to subside can passive motion be cautiously begun, and not until still later can any form of local stimulation or voluntary activity be safely ventured on.

An affected limb should be wrapped in a generous quantity of cotton secured by a bandage just tight enough to stay in place. The limb should be in the posture of greatest ease. The arm is to be moderately abducted at the shoulder, the elbow slightly flexed, the forearm between pronation and supination, the wrist slightly extended, and the fingers moderately flexed. The lower limb is to be slightly flexed at the hip and knee with a little outward rotation and abduction, the foot being at right angles to the leg. The proper posture is to be maintained by the support of soft pillows of suitable size so that both muscles and nerves will be relaxed and free from pressure, with no occasion for voluntary effort. Wrist drop or foot drop may be prevented from the first by a proper arrangement of the supports. For the feet a large sandbag nine inches in diameter is desirable, placed transversely in the bed so that the ball of the foot and toes will rest upon it in such a way as to secure the perpendicular position (Gowers, 12). Or a board may be secured across the bed for the feet to rest upon. The weight of the bed coverings must not rest upon the limb. If the supports mentioned do not sufficiently protect it a wire cradle must be used.

It is best to maintain an equable warmth of the affected part. Changes of temperature prevent sensory rest, hence they are to be avoided as carefully as mechanical irritation. In the most acute cases a warm, moist dressing or poultice gives more relief than the dry cotton. Especial care must then be taken to see that the temperature is below what can be borne with comfort by the sound skin, on account of the extreme vulnerability of tissues supplied by an inflamed nerve and the consequent danger of severe trophic disturbances. Warmth with some moisture may be secured by using cotton as recommended above, but covering it with rubber tissue. Some writers recommended cold applications in severe local inflammation, but it must be remembered that the tissues are excessively vulnerable to cold as well as to heat and pressure. In general, warmth is to be preferred.

In recent cases, especially if severe, massage and electricity are strictly contraindicated, but are often urged upon the physician by the patient or his over-zealous friends. Even the electric tests that are desirable for diagnosis to some extent aggravate the disease, as indicated by increase of pain, so they should at first be as brief and infrequent as the diagnostic requirements will permit. I have known patients with alcoholic multiple neuritis to have their sufferings greatly increased and the prospects of recovery lessened by daily applications of a strong faradic current, the physician as well as the patient being under the delusion that the increased pain following each application ought to be endured because electricity may be useful in the treatment of paralysis.

Relief of Pain.—The removal of causes and the maintenance of rest in the easiest posture tend strongly to relieve pain, but, in spite of all that can be done in this way a good deal of neuritic pain will usually remain, and it is of such a peculiarly trying, nerve-racking character as to prevent refreshing sleep and seriously to impair the general health. Additional measures for the relief of pain alone are generally necessary, but Gowers wisely warns against using these measures to permit harmful activity, which, without analgesics, would be too painful. In other words, analgesics and narcotics should be sparingly though efficiently used for the relief of the spontaneous pain which persists after the most perfect rest and protection have reduced it to a minimum.

Gowers (12) strongly recommends the hypodermic injection of cocain at the seat of greatest pain, not only as an efficient though temporary means of relieving suffering, but also as having a distinctly favorable effect upon the morbid process by cutting off irritating sensory impulses from the periphery. Oppenheim (30) also mentions it as a palliative, especially in the form of Schleich's infiltration anesthesia. From one-tenth to one per cent. of cocain in normal salt solution is employed and injected near the nerve in the area of greatest pain. At first not more than gm. 0.005 or 1-12 of a grain of cocain should be used. When the patient's tolerance is known as much as gm. 0.065 or one grain may be injected twice daily. The weaker the solution the larger the area that can be covered without exceeding the dose. Gowers also speaks of cocain satisfying the craving for a stimulant, but this also carries with it the warning that a craving is easily created by the use of the drug and that the cocain habit is peculiarly demoralizing. These objections to cocain may to a great extent be obviated by substituting one of its less poisonous substitutes, eucain, stovain, novocain or alypin, in the same or larger doses. The lately introduced local anesthetic, hydrochlorid of quinin and urea, is said to be entirely harmless and to cause an analgesia which persists for days. I have had no personal experience with it, but should the claims for it be confirmed it would be far more valuable for this purpose than any drug of the cocain class.

As an internal analgesic, unless contraindicated by the state of the heart or of the blood, one of the coal-tar preparations should be tried. Acetphenetidin (phenacetin) is probably the best, and may be given in single doses of from gm. 0.3 to 1.0 (5 to 15 grains), maximum in one day gm.

2.0 (30 grains). Instead of acetphenetidin one of the similar drugs may be used in its appropriate dose: kryofin in the same doses as acetphenetidin; antipyrin, gm. 0.65 to 2.0 (10 to 30 grains), maximum in one day, gm. 4.0 (60 grains); salpyrin in the same doses as antipyrin; acetanilid, gm. 0.2 to 1.0 (3 to 15 grains), maximum in one day, gm. 1.50 (23 grains), pyramidon in the same doses as acetanilid.

If the coal-tar preparations are contraindicated or ineffectual an opiate will be necessary. Codein is nearly harmless and may be given in a single dose of from gm. 0.03 to 0.15 (½ to 2 1-3 grains). Purified opium has more power to relieve pain and also to secure sleep, doses gm. 0.03 to 0.13 (½ to 2 grains), or extract of opium, gm. 0.015 to 0.07 (¼ to 1 grain); or morphia, gm. 0.01 to 0.03 (1-6 to ½ grain). If one of these is not sufficient hypodermic injections of morphia will be necessary, especially at night. All authorities are agreed that this should be only as the last resort. Unless the patient's tolerance is already known the first dose must always be small, then the amount can be cautiously increased to meet the necessities of the individual case.

Sleep.—It is of great importance that a patient suffering from neuritis should have sound and refreshing sleep. The opiates made necessary by pain may be sufficient to secure this, but it is often desirable to add one of the hypnotics.

I regard chloral as on the whole the best hypnotic. It will generally secure sleep of the necessary duration, and of a refreshing quality, with less disadvantages than any other single drug. Its dose is from gm. 0.5 to 1.5 (7½ to 23 grains); maximum in one night, gm. 2.0 (30 grains). Bromid may be used instead in a single dose of gm. 1.0 to 2.0 (15 to 30 grains), but is not so efficient, and larger doses, if continued, seem to me more likely to depress nutrition and lower resistance to infection than equally efficient doses of chloral. Veronal is very efficient in inducing sound sleep. The principal objection to it is that it often leaves the patient unrefreshed, heavy, and rather despondent next day. When it acts in this way its prolonged use is highly objectionable. Some patients, however, bear it very well, and with them it is the hypnotic to be chosen. Whatever is necessary should be given in a single dose of from gm. 0.3 to 1.0 (5 to 15 grains), as its maximum effect is some hours after its administration. The solution of veronal known as neuronidia has the advantage of acting with the minimum dose in the shortest time.

Counterirritation.—In multiple neuritis counterirritation is not advisable, but in mononeuritis due to a sharply localized injury or infection it may be of use. Light stroking with the Paquelin cautery over the nerve a little above the seat of inflammation is most effective, but small blisters may be used instead. Such applications are not to be made in the distribution of the nerve below the seat of disease for fear of starting an ulceration which may persist as a trophic lesion. It is in the later stages of the disease that counterirritation is most useful, but in milder cases it may be used at the outset. In the early stages of severe cases it is best to omit it.

Massage.—As spontaneous pain subsides and that induced by passive motion of the affected part is of short duration and less intense, massage, with systematic passive motion, may safely be begun. This may be in a few days in the lightest cases, and in from four to eight weeks in the severe ones. In the meantime all that is necessary in the line of mechanotherapy is the support of the foot and hand that will prevent foot drop and wrist drop.

The object of massage is to accelerate the flow of lymph and venous blood from the diseased tissues so that fresh lymph and blood may take their place. This is to be accomplished with the least irritation and the nerve trunk must be avoided. At first the manipulations should be exceedingly gentle, consisting only of a light rubbing or stroking of the skin in the direction of the venous current. As tenderness subsides the stroking may be somewhat more vigorous and enough pressure may be used to influence the deeper tissues. Severe or persistent pain following massage should always cause it to be made lighter or for the time to be omitted altogether. With the massage gentle passive motion of all the joints should be made, carefully avoiding all brusque changes and extreme positions. At the same time the posture should be varied from that of greatest ease so that the patient will become accustomed to lie with both the hip and knee extended.

Electricity.—All authorities are agreed as to the value of electricity in the treatment of neuritis; and electrical treatment should be begun soon after the first use of massage and passive motion. It is also generally agreed that the galvanic current is superior to all the other forms of electricity. The faradic current is valuable for diagnostic tests, but in this disease its power to cause contraction of the affected muscles is lost or impaired, while its effect on the inflamed nerve is that of a powerful irritant. In chronic or convalescent cases a neurologist who is also an expert electrotherapist may do good with it, but even then more can be accomplished through galvanism. I think that it should be a general rule not to use faradism in the treatment of neuritis.

The galvanic current is used in two ways for two distinct purposes. As originally recommended by R. Remak and endorsed by E. Remak, Bernhardt (2), and Oppenheim (30), the cathode is applied over the seat of the nerve lesion and the anode to any convenient place. The current is turned on gradually by means of a smoothly working rheostat, kept steadily at a strength of from two to six milliampères for from five to ten minutes, and then gradually turned off. All abrupt changes must be avoided. The weaker and briefer applications are best at the beginning and in all cases in which the nerve is excessively sensitive; as the effect of the applications is ascertained and sensitiveness is found to be decreasing, currents may be used for a longer time and their strength cautiously increased. If severe or persistent pain is caused the current must be decreased or the treatment postponed. Gowers (12) employs a similar method, but he uses the anode over the inflamed part and suspects that its value is only that of a counterirritant. According to the German neurologists the effect of these applications is to improve circulation in and about the inflamed nerve, to promote the absorption of the products of inflammation, and to improve the conductivity of the nerve fibers by direct stimulation. E. Remak gives careful observations tending to prove that these things are really accomplished and the course of the disease materially shortened. The results obtained by others generally confirm his claims; certainly the galvanic treatment of the nerve itself in old cases of moderate severity is often followed by a striking improvement after only a few applications.

The other method is to apply the cathode or anode, whichever causes the greater contraction, to the paralyzed muscles without regard to the nerve, and by slowly making and breaking the current directly to stimulate the muscle to contract. Its object is to exercise the muscles and keep them in the best possible state of nutrition, delaying their degeneration until they come under the influence of the restored motor fibers and end plates. This direct stimulation to contraction is just what the faradic current cannot accomplish, because it acts on the muscles only through the nerve fibers, which are destroyed or put out of action by the disease. Although this galvanic treatment of the muscles is not necessary in the lightest cases, and is of little avail in the most severe ones, where the restoration of the nerve fibers is long delayed and the muscles lose even their galvanic reactions, nevertheless there is a great preponderance of cases in which this treatment is eminently useful. It should be tried in practically every case. The applications may be from three times weekly to daily. Each muscle is to be relaxed by posture so that it is free to contract without resistance. The strength of the current will vary from two or three to ten milliampères according to the muscle treated and the size of the active electrode. It should be just sufficient to cause a moderately vigorous contraction. Very strong currents are to be avoided as they lead to exhaustion.

Baths.—In the middle and later stages of the disease warm baths may be used to favor relaxation, peripheral circulation, and absorption.

Exercise.—As soon as some voluntary control of the paralyzed muscles is regained active exercises should be added to the passive motions already mentioned. The weakened muscles should at first be favored by posture and by the assistance of the operator, so that the patient's efforts may produce the greatest visible result. Care is to be taken to stop short of severe fatigue.

Late Operations.—If, after more than a year from the onset, there is still considerable disability, with but little hope of further improvement, careful consideration should be given to the possibility of a late performance of neurolysis or nerve anastomosis leading to better results. Orthopedic operations such as section of tendons or transplantation of muscles may possibly be indicated, but not so frequently as in poliomyelitis. Orthopedic apparatus may also be useful without operation.

Drugs.—Aside from the treatment of causes such as syphilis, malaria, or metallic poisoning, drugs can have but little effect upon the morbid process. Gowers, indeed, speaks highly of mercury as an alterative in localized neuritis, one grain of blue pill once or twice a day. He does not consider it so useful in multiple neuritis. Salicylate of sodium given as in acute rheumatism is useful in cases due to cold or having a rheumatic or gouty element. But if drugs can do little to cure the neuritis itself, they are valuable for the relief of many symptoms. The relief of pain and insomnia has already been discussed. Throughout the course free elimination must be maintained, especially by the judicious use of laxatives. Tonics, especially strychnia and iron, will be needed in all protracted cases.

Change of Scene.—As in other long illnesses convalescence may often be hastened by a change of air and scene, especially if agreeable recreation accompanies the change. In winter the patient should go to one of the warm, balmy localities, in summer to the seashore or mountains, but never to regions that are both cold and damp.

NEURITIS OF SPINAL NERVES

1. Phrenic Neuritis

(*Paralysis of the Diaphragm*)

Only very rarely is disease of the phrenic nerves causing paralysis of the diaphragm amenable to special treatment. When the nerve is involved in wounds, inflammation, or tumor of the neck the treatment, if possible, of the primary disease is the essential object. When inflammation of the phrenics is a part of multiple neuritis their treatment is for the most part included in that of the greater disease. The danger is especially great on account of the dyspnea and danger of complete failure of respiration. Extreme care should be taken to secure the most perfect rest possible. Talking as well as other forms of exertion is to be prevented. In order that the abdominal contents shall not encroach upon the thorax the head and chest should be elevated. If dyspnea persists in spite of complete rest and correct posture inhalations of oxygen may be used. Counterirritation along the sides of the neck has been recommended and no doubt is useful in cases of active inflammation. Strychnia and an abundance of food are indicated.

In addition to these measures Duchenne recommended faradization of the nerve, and this has been warmly approved by Bernhardt (2), and briefly by Oppenheim (30). A small button-shaped electrode is applied at the posterior border of the sternomastoid muscle, at the junction of its middle and lower thirds, and pressed downward and inward between this muscle and the scalenus anticus, while the other electrode is applied over the abdomen. When the nerve is healthy the diaphragm can easily be made to contract in this way. Gowers (12) thinks that the influence of this procedure is not sufficient to make its use desirable, and he seems clearly to be right as far as disease of the nerve trunk is concerned. If the nerve fibers are diseased neither the faradic nor the galvanic current applied in this way can have much effect. In the cases of asphyxia from gas poisoning and of apparent death in the newborn it is not the nerve fibers, but the nerve centers, that are probably at fault, and artificial respiration seems to me to be far more promising than faradization.

As to prophylaxis, care should be taken by anesthetists, surgeons, and obstetricians not to turn the head too forcibly to one side and not to keep it long in an extreme position. Oppenheim has seen paralysis of one phrenic nerve caused by such forcible turning.

2. Neuritis of the Brachial Plexus and Nerves of the Arm

In neurological treatises the diseases of the parts of the brachial plexus and of the nerves arising from it are discussed in separate chapters. This separation, however, is on account of varying symptomatology and for the sake of localization diagnosis, not on account of difference in treatment. For the discussion of treatment alone and especially to avoid tedious repetition it seems better to consider them together.

The brachial plexus and the nerves derived from it are not only liable to the same toxic and infectious diseases as other nerves, but are especially liable to disease and injury incident to their situation. The use of the arm exposes it to many special dangers. Blows, cuts, punctures, and pressures on the unprotected soft parts of the arm or neck often involve the nerves, as a rule directly, sometimes by subsequent extension of disease. Fractures and dislocations often cause pressure or laceration of the plexus or the nerve trunks, especially the musculospiral. Moreover the great mobility of the bones of the shoulder and arm permits a number of extreme postures, without either fracture or dislocation, which are capable of causing great injury to the nerves, amounting in some cases to a complete separation of nerve roots from the spinal cord. These facts make it necessary to give special attention to prevention.

Prevention.—A great many cases of brachial neuritis could be prevented if the possibility of their occurrence were generally known. A statement of some of the special causes is sufficient indication for their prevention.

A workman carries a heavy weight on the shoulder so that its edge presses into the side of the neck; pressure neuritis of the posterior thoracic nerve and paralysis of the serratus magnus muscle follow, or perhaps neuritis of the upper cord of the plexus and paralysis of the upper arm type of Erb. The shoulder is forcibly drawn back as in pinioning the arms; the upper cord of the plexus is squeezed between the clavicle and first rib so that paralysis of the upper arm type follows. A crutch is used so that the weight of the body, instead of being supported mainly by the hands, is borne by the axilla; neuritis of the musculospiral results with paralysis of the extensors of wrist and fingers. Severe strain on the uplifted or extended arm, as in hanging from a ladder or reining an unruly horse, stretches the upper part of the plexus, especially if the head is inclined away from the arm. Other easily avoidable causes are allowing the arm of an anesthetized person to hang over the edge of the table, holding the arms above the head for a considerable time, going to sleep with the arm hanging over the back of a chair, lying with the head resting on the upraised arm, merely the prolonged maintenance of an awkward position and excessive work with the arm raised above the head, as in whitewashing a ceiling. Tight bandaging, or the prolonged action of a bandage that is even a little too tight, may cause severe neuritis. In giving hypodermic injections a nerve cannot be injured unless the deep fascia is pierced; this can be avoided by pinching up a fold of the skin.

According to Goldthwait, Painter, and Osgood (11), neuritis of the ulnar nerve may be caused by the common deformity, round shoulder. As the shoulder joint, from fatigue and relaxation of the muscles which normally hold it backward and upward, sags downward and forward, the head of the humerus may compress the ulnar nerve against the second rib, causing a true pressure neuritis. In thin persons the ulnar nerve alone is usually affected, but in stouter patients a padding of fat may transmit the pressure to the whole plexus. The pain thus caused is aggravated by postures and occupations which increase the forward drag on the shoulder and relieved by postures which elevate the head of the humerus and keep it away from the thorax. Some cases of writers' cramp are to be explained in this way. The treatment consists of exercises and a brace to hold the body erect and the shoulders well back. If the scapula is flexed so that the correct posture is painful and the patient is an adult, removal of its upper portion may be necessary.

Obstetric Paralysis

The prevention of injury to the plexus or nerves at birth which causes the paralysis, usually of the upper arm type, known as obstetric paralysis, is a special problem for the obstetrician, but the same general principles apply. Delivery is to be accomplished, avoiding as far as possible any extreme position of head or arm and any excessive traction on either, especially traction on the arm when the head is inclined away from it. Any prolonged pressure is to be feared, especially if on the neck, shoulder, axilla, or arm.

Treatment.—In traumatic cases prompt aseptic dressing of wounds and immediate treatment of a fracture or dislocation are most essential. The question whether any nerve trunk is completely divided comes up at once and is answered mainly by the presence or absence of sensation in the area supplied by the nerve in question. The ability voluntarily to contract the muscles supplied by the nerve, of course, shows that the nerve is not divided, but the inability to make such voluntary motions is not proof of division of the nerve because it may be due to injury of bone, muscle, or tendon. If division has occurred immediate suture is vastly better than secondary suture, but no limit can be set beyond which operation is hopeless. If there is a large gap in the nerve trunk resection of the humerus may be justifiable as a means of bringing the ends together. If the gap is too great to be closed in any way the peripheral part of the severed nerve may be grafted into a neighboring sound one, as the musculospiral into the median. If a fragment of bone or callus is pressing on the nerve it must be removed. Callus may first cause paralysis years after the original injury (Oppenheim, 30). If the nerve is embedded in inflammatory products or drawn by contracting scar tissue it should be exposed by careful dissection beginning either below or above the lesion, freed from adhesions, and protected from future ones, if possible, by placing it in a more favorable situation between the muscles or wrapping it in a flap of fascia. The ulnar nerve is sometimes dislocated from its position back of the inner condyle of the humerus. On account of its great liability to injury in its new location it should be replaced and retained by covering it with a flap of periosteum (Chipault, 6).

After pain and other acute symptoms have sufficiently subsided the absolute rest which is at first imperative should gradually be broken by passive movements, massage, and galvanic electricity, as described under the treatment of neuritis in general. The galvanic current should be applied in the two ways already mentioned, that is, smoothly and steadily with the active negative electrode over the lesion and the anode on the muscles, and also slowly interrupted with the active electrode on the muscles, whichever one gives the greater contraction.

In the obstetric paralysis of infants galvanic treatment should be begun early and, if necessary, be persisted in for many months. If at the end of a year there is no prospect of further recovery under this treatment the injured part of the plexus or nerve should be exposed, freed from surrounding scar tissue and, if necessary and possible, resected and sutured. For details see the articles of Clark, Taylor, and Prout (7).

If, after all attempts to restore nerve function, an important muscle remains paralyzed, the condition may perhaps be improved by splitting off a portion of a sound neighboring muscle and attaching it to the insertion of the paralyzed one. Thus a part of the sound pectoralis major has been stitched to the paralyzed serratus magnus or deltoid; part of the deltoid or biceps to the triceps; the sound flexor carpi ulnaris to the paralyzed extensors of the wrist and fingers, etc. These are highly specialized operations whose scope and value are still under discussion and vary greatly in the opinions of those best qualified to judge. They should not be undertaken except by those who have special knowledge and skill in this particular field.

3. Neuritis of the Lumbar Plexus and Its Distribution

a. External Cutaneous Neuritis

(Meralgia Paresthetica)

This affection, limited to the external cutaneous nerve and causing a painful numbness of the skin of the outer side of the thigh, is rare but of considerable importance in some cases, so that it has been made the subject of a number of special articles. Its commonest causes are infections (especially typhoid fever, syphilis, and articular rheumatism), injury, exposure to cold, alcoholism, obesity, and diabetes.

Bernhardt (3), who first described it, says that a very important part of the treatment is the assurance of the alarmed patient that the disease is limited, not progressive and not serious. Treatment should naturally be directed to whatever cause is discovered, as prevention of pressure by the sword worn by officers, abstinence in alcoholism, mercury in syphilis, salicylates in rheumatism, diet in diabetes. Analgesics, counterirritation, and electricity may be used as in other forms of neuritis. While often obstinate, the disease is not as a rule serious enough to demand operation. The nerve, however, can very easily be resected or, better still, injected with alcohol where it passes beneath Poupart's ligament, close to the anterior superior iliac spine. Neisser and Pollack (25) regard the disease as caused by pressure on the nerve by the margin of the fascia lata and have advised slitting the fascia.

b. Anterior Crural and Obturator Neuritis

Localized neuritis of these nerves is rare, but the possible causes are numerous. The most important of these are new growths (starting from the spine, retroperitoneal glands, pelvis, or femur), psoas abscess, appendicitis, aneurysm of the femoral artery, cold, gout, alcoholism, diabetes, injury to the pelvis or femur, and prolonged flexion of the hip during a surgical operation. After removing causes as far as may be possible the general treatment for neuritis should be employed. If further restoration is not to be expected a flexor tendon, that of the biceps for example, might be attached to that of the quadriceps extensor, as has been done after poliomyelitis (Oppenheim).

c. Sciatic Neuritis

(Sciatica)

The term sciatica is commonly understood to mean pain of a neuralgic character in the course and distribution of the sciatic nerve, without the symptoms that would indicate neuritis or other organic disease. Gowers thinks that the great majority of cases described as sciatica are really cases of neuritis, as shown by the character of the pain, more or less sensory loss, absence of Achilles jerk, and wasting of the muscles. Oppenheim (30), on the other hand, holds that the indications of neuritis are generally very slight and that most cases are of a neuralgic rather than of a neuritic character. Accordingly he describes sciatica as a neuralgia separately from sciatic neuritis. He says, however, that it is impossible always to distinguish the two, and that every possible gradation exists between a manifest neuritis and a pure neuralgia. As the treatment is for the most part the same, and as I regard sciatica as neuritic rather than neuralgic, all forms are considered here, with no attempt to differentiate them except as to their causes.

Fully developed sciatica is a very formidable disease. The disability and severe suffering caused by it and the obstinate resistance of many cases to a great variety of modes of treatment have made it, like trigeminal neuralgia, a reproach to our profession. The main reason for this lack of success in the difficult cases is not that these cases, like those of tabes, are essentially incurable. It is because the causes of the disease are so many and often so obscure that in a given case it is very difficult for the physician to determine what is the fundamental indication for treatment. Removal of any active cause is essential, yet this is often left to nature while efforts are concentrated on the obvious necessity of relieving pain. As an unknown cause sometimes disappears spontaneously, but often does not, merely palliative treatment may be brilliantly successful in one case and a total failure in another. Hence the great number of remedies recommended and the consequent confusion and uncertainty. This is further increased by reporting as sciatica, cured by this or that treatment, cases of pain, referred to the region of the sciatic nerve, which is neither neuralgic nor neuritic, but neurotic, that is hysterical. These may be converted into cases of grave organic disease by mistaken treatment. Oppenheim mentions a case of irremediable peroneal paralysis caused by exposing the nerve and applying strong carbolic acid to it to cure a sciatica which was hysterical. Under the rules of a hospital requiring consultation before major operations a neurological colleague and myself were once called upon to approve an amputation of the thigh for which an hour had been set. The patient's complaints of pain had led to a section of the peroneal nerve. This giving no relief a piece of the nerve two inches long was removed at the level of the head of the fibula. The complaints continuing, amputation was proposed and accepted by the patient. We found no evidence of any organic disease or any form of sciatica, but regarded the pain as clearly hysterical. The amputation was not done, but the complete wasting and paralysis of the anterior tibial and peroneal groups of muscles of course remained.

Assuming the purely neurotic imitation of sciatica to be excluded we have to remember that the fibers of the nerve in their long course from the spinal cord, at the level of the first lumbar vertebra, through the spinal canal, pelvis, thigh, and leg are exposed to many possibilities of damage through localized injury or disease. A thorough examination from both the neurological and surgical standpoint is necessary in order to detect or exclude disease in each of the various parts of its course. For the purpose of treatment our best classification is one based on causation, first local, then general.

Radicular Sciatica.—The roots of the sciatic nerve are long, reaching from the level of the first lumbar vertebra to the fifth lumbar and first three sacral intervertebral foramina. They are liable to damage by meningitis, especially that caused by syphilis, fractures of the vertebræ, inflammation of the vertebræ, especially Pott's disease, new growth of the bones or meninges, and arthritis deformans encroaching on the intervertebral foramina. In radicular sciatica the pain is commonly bilateral, and may also be felt in the distribution of the anterior crural or other lumbar nerves. The tender points of Valleix and the sign of Lasègue (aggravation of the characteristic pain of sciatica by extending the leg when the thigh is at right angles to the trunk) are absent. The pain is increased by coughing. Sometimes areas of hyperesthesia corresponding to those supplied by definite roots may be made out.

The treatment of this form of sciatica is not often successful because the original cause is generally beyond cure. If there is a suspicion of syphilis mercury should of course be tried, and followed by iodids. Pott's disease may yield to proper mechanical support and constitutional treatment. For the relief of pain the epidural injections of Cathelin should be tried, and are much more likely to succeed than injections applied to the trunk of the nerve in the buttock or thigh. After sterilization of the skin with alcohol ether and tincture of iodin, and placing the patient in the knee-chest position, or on the side affected with the thighs flexed, the needle, such as is used for lumbar puncture, is introduced in the median line of the spine, between the sacral tubercles and 7 to 7.5 cm. (2 4-5 to 3 inches) from the tip of the coccyx. It is introduced perpendicularly until the sacrococcygeal ligament is pierced, which can be recognized by the feeling of piercing a membrane, when the needle is pointed upward at an acute angle, so as to enter the sacral canal without penetrating the dura. The depth of penetration is 4 to 5 cm. (1 3-5 to 2 inches). Two c. c. of a one per cent. solution of cocain or two to four c. c. of a one half per cent. solution of stovain properly sterilized are to be slowly injected. A characteristic sensation is generally felt, formication in the limbs and a "feeling of distention spreading upward from the buttocks, or of water flowing in the loins." The pain is promptly relieved and the relief may last two or three days, perhaps ultimately becoming permanent (Lannois and Porot, 21). Lévy and Baudouin (22) recommend larger injections, gm. 0.01 or 0.02 (1-6 to 1-3 gr.) of cocain, or twice as much stovain, in 20 c. c. of normal salt solution, but they say that epidural injections in radicular sciatica are inconstant in their effect, which is to be expected in a disease of its varying causation. If these injections fail they recommend subarachnoid injections, as in spinal anesthesia for operations, but with smaller doses of cocain or stovain, not exceeding gm. 0.03 to 0.04 (½ to 3-5 of a grain). The relief, of course, is generally only temporary, and this method is not recommended for ordinary sciatica.

Sacroiliac Sciatica.—One of the most important recent advances in the knowledge of sciatica is the discovery that many cases are caused by dislocation, sprain, relaxation or inflammation of the sacroiliac joint, and that these cases can be cured only by measures which will give the joint rest and support. The lumbosacral cord, whose continuation forms the peroneal nerve, and the first sacral nerve are immediately in front of the joint, while the second and third sacral nerves are separated from it only by the origin of the pyriform muscle. Swelling of the joint or displacement of the upper portion of the sacrum either forward or backward stretches these nerves. I have seen the most intense and characteristic sciatic pain instantly caused for the first time by a sprain of this joint, brought about by a misstep in the dark. Aside from peculiarities in standing and walking, which might be attributed to sciatica from any cause, disease of the sacroiliac joint causing sciatica may be recognized by the following tests, abstracted from the book of Goldthwait, Painter, and Osgood (11), to which the reader is referred for a full explanation: 1. Standing with the knees extended while bending the trunk forward as though to pick up an object causes pain in the affected joint and lumbar region, with reflex spasm of the lumbar muscles restricting the motion. This is because the pull of the hamstring muscles on the ischium prevents the pelvis from tilting forward, so the sacrum must move on the ilia, causing a strain of the sacroiliac joints. If the patient now sits with the knees flexed and bends forward the pain and restriction of motion are much less, because the hamstring muscles being relaxed the ilia are free to tilt forward with the sacrum, putting but little strain on the sacroiliac joints. Were the disease in the lumbar spine, as the location of the pain and spasm might lead one to suspect, forward flexion would be equally painful and difficult whether standing or sitting. 2. If the patient lies on the back, and the thigh on the affected side is flexed on the trunk, with the leg extended, as in the familiar test for sciatica, pain is felt not only along the nerve, but in the sacroiliac joint and lumbar spine. This test is the converse of the preceding, the hamstring muscles moving the ilium on the sacrum. If the same manipulation is done on the sound side pain is felt on the affected side, but it is less severe because the pull of the hamstring muscles first moves the ilium of the sound side on the sacrum, which is then moved on the ilium of the affected side. If in making this test the thigh is flexed on the trunk with the knee also flexed but little pain is felt, except in extreme positions or in exceedingly acute cases. This is because the hamstring muscles are relaxed and do not pull on the ischium. In hip joint disease flexion or extension of the knee would make no difference. 3. If the joint is inflamed swelling and tenderness may perhaps be recognized by palpation from without or by rectal examination. 4. Increased mobility of the joint may be recognized by the examiner, palpating the sacroiliac joints with one hand and the symphysis pubis with the other, while the patient flexes either thigh with the knee extended. This will also cause the characteristic pain and reflex restriction of motion as in tests 1 and 2.

If this joint is dislocated, the dislocation must, of course, be reduced and a dressing applied to maintain the bones in place. If strained and inflamed the first requisite is rest, which in all severe cases should begin with rest in bed. The joint should be fixed by a plaster jacket, taking in the thorax, the hyperextended lumbar spine, the pelvis, and, if necessary, by a spica extension, the thigh or even both thighs. After acute symptoms have subsided, and in milder cases from the first, support of the joints may be all that is necessary without confining the patient. This support may be a plaster jacket or a belt firmly girding the pelvis between the iliac crests and the trochanters, and held down by perineal straps or attachment to the corset. At night a small pillow under the lumbar spine and one under the knees is of special importance. In mild cases, for temporary relief, overlapping strips of adhesive plaster, carried from the anterior part of the ilium on one side to the corresponding part on the other, may be sufficient. For important details the reader is referred to the work of Goldthwait, Painter, and Osgood (11), to Pitfield (34), and to Young (44).

The orthopedic indications having been fulfilled, this form of sciatica has a strong tendency to get well without other treatment. Pain may for a time require relief, and a diathetic condition, such as rheumatism, may call for treatment. Injections into the nerve trunk are not indicated in this form of the disease.

Sciatica from Intrapelvic Disease.—Within the pelvis the sacral plexus may be injured by various processes; an infection may extend to the nerves from any of the pelvic organs, an exudate or a new growth may encroach upon them, and harmful pressure may be exerted by the fetal head, by the enlarged or displaced uterus, by a fecal accumulation, or even by venous congestion. The pelvic examination must be most thorough, and, if any of the conditions mentioned is found to have a causal relation to sciatica, its successful medical or surgical treatment is essential. In cases where suppuration within the pelvis has affected the nerves, in addition to the ordinary methods of drainage, an appropriate vaccine should be used.

Sciatica from Damage to the Nerve Trunk.—In its course through the thigh and leg the sciatic nerve may be exposed to many causes of mechanical damage or local infection, such as the pressure of a hard seat; injuries, inflammations, or operations about the hip or knee; fractures, especially of the upper part of the fibula; violent muscular action or working in a kneeling posture. The detection of any of these causes will carry with it the indications for the treatment necessary to relieve the nerve of pressure, irritation, or continued infection. If the nerve trunk is believed to be enveloped in exudate or bound down by adhesions, it should be exposed by dissection and freed. This is decidedly preferable to the so-called "bloodless stretching." The nerve will almost always tend toward recovery, however slowly, as soon as the original cause is removed.

Sciatica from Constitutional States.—If no cause of local damage to the nerve roots, plexus, or nerve trunk can be discovered, a constitutional cause will probably be found. The sciatic nerve, especially its peroneal part, like the musculospiral, is excessively vulnerable in the presence of a general infection or intoxication. There is no specific infectious disease which may not exert a selective influence upon the peroneal nerve so as to cause neuritis with the accompanying pain, paralysis, and atrophy. Puerperal fever, typhoid, influenza, even gonorrhea may be such a cause. Of the intoxications, alcoholism, gout, diabetes, and poisoning by arsenic and lead must be considered. Exposure to cold is a very important cause. These are also the causes of multiple neuritis, but they may act, with or without slight local injury, so as to cause sciatic neuritis alone; or sciatica may persist after the other nerves involved in multiple neuritis have recovered. In any case of sciatica, therefore, even though a local cause may be apparent, these possible constitutional factors must be looked for, and, if found, efficiently treated. Gout or muscular rheumatism will be found more frequently than any other one constitutional cause, and salicylates with saline laxatives will be found more generally useful than any other drugs.

[Chronic intestinal autointoxication should always be looked for, especially in the recurring form.—Editor.]

Symptomatic Treatment.—In any case of sciatica, while causes are being removed as far as possible, and, indeed, after they have been removed, special treatment of symptoms will be necessary, for if once damaged the nerve recovers but slowly.

The first essential, as in other cases of neuritis, is rest, in the posture of greatest ease. In all severe cases the patient must be in bed, the limb being supported by pillows so that the thigh is slightly flexed and rotated outward and the knee slightly flexed. The foot is to be kept at right angles to the leg, by large sand bags, or a board placed across the bed, and must be protected from the weight of the bedclothes. Some support under the lumbar spine to maintain its normal curvature is also advisable.

Cold applications are generally to be avoided, because disagreeable and likely to depress the tissues. Warmth, on the other hand, is soothing and useful. Hot fomentations may be applied by Siegrist's method, described by Oppenheim (30) as follows: "A towel folded lengthways, so that it forms a compress 10 cm. (4 inches) broad, is immersed in hot water of 40° to 50°R. (122° to 144° F.), wrung out, and laid along the affected nerve; over this is placed a broad strip of flannel which covers the towel, and above this again several layers of paper"; all held in place by a broad roller bandage. "The fomentation is renewed after ten to fifteen minutes, and this process is kept up for one to two hours several times a day." Lévy and Baudouin (22) recommend full warm baths lasting an hour. Cupping, wet or dry, and the application of leeches have been found useful.

Counterirritation may be used in the form of friction with irritating liniments, mustard leaves, repeated small blisters along the course of the nerve, or, most efficient of all, light stroking with the Paquelin cautery over the seat of the greatest tenderness. The faradic brush and static spark have been recommended as counterirritants, but have no advantage to make up for their inconvenience unless it be a mental impression.

The galvanic current is useful; large electrodes should be employed and the current can be in proportion to the area of the active one, ¼ to ½ a milliampère for each square centimeter, or 1½ to 3 milliampères for each square inch. The active cathode should be over the seat of greatest tenderness, and the anode over the lower course of the nerve. The current must be turned on and off gradually. In most cases of sciatica the muscles are not affected so severely as to need treatment to keep up their nutrition, but, if they are paralyzed, they should be stimulated to contract by means of the galvanic current, as described in the treatment of neuritis in general. What has there been said in regard to massage also applies here.

Injections Into the Nerve Trunk.—For prompt relief of pain and also for ultimate cure, in properly selected cases, a method of treatment by injection into the nerve sheath has been developed, mainly within the past six or seven years, which gives far better results than any of the earlier modes of treatment. It is especially valuable, as it is most successful in the cases in which the causes are obscure and rational treatment has accordingly been most difficult.

Cases Benefited by Injections.—The cases most likely to be cured or greatly benefited by injections are the subacute and chronic ones, not due to disease of the spine, sacroiliac joint or pelvis, in which pain persists after all known causes have, as far as possible, been removed. If the cause is either spinal or pelvic the epidural injections of Cathelin, already described, are more efficient, because they act on the nerve roots above the seat of irritation. Injections into the nerve trunk are not so strongly indicated in the acute cases, in which other measures would naturally be given a trial first. Nevertheless, they have been successfully used in a considerable number of acute cases. Hysterical patients with pseudo-sciatica should not receive this treatment; it may happen to make the right mental impression, but this can better be secured in simpler ways.

Solution for Injection.—Cocain, eucain, stovain, novocain, or any other of the local anesthetics, if injected in the usual quantities of a hypodermic injection into the nerve or very close to it, *above the seat of irritation,* will give complete relief for a short time, usually not more than a few hours. If injected into the nerve trunk when the irritation is in the pelvis or spinal canal, some relief will be obtained, because the diseased part is shielded from the additional irritation of impulses from the periphery, but the relief will naturally be incomplete as well as temporary. The great success of the alcohol injections introduced by Schlösser in 1904 for trigeminal neuralgia naturally led to their trial in sciatica. Here, too, they at first promised to be highly successful. Nevertheless, alcohol for this purpose has been entirely abandoned, because it is capable of destroying the conductivity of nerve fibers, and, as the sciatic is a mixed nerve, with most important motor and trophic functions, there is too great a risk of causing a complete paralysis and wasting of the muscles supplied by it. It was at first thought that alcohol would not cause such a paralysis, and in the early cases of Schlösser and his followers it did give relief from pain without paralysis, but later cases and many experiments on animals show that, if the alcohol is strong enough to relieve the pain and is really injected into the nerve trunk, it will almost inevitably cause an atrophic paralysis, which may be many months in disappearing, if it disappears at all. The use of alcohol or any other destructive agent, such as chloroform, osmic acid, or carbolic acid, is not justifiable, especially as there are solutions which are both safe and efficient.

It is now generally agreed that the salt solution introduced by Lange is harmless, or nearly so, and that it or some modification of it should be chosen. A large quantity should be used, 40 to 100 c. c. (1 1/3 to 3 1/3 fluid ounces), or even more; 60 c. c. (2 fluid ounces) is an average amount. Many variations of the solution have been used, some preferring strongly hypertonic, others isotonic salt solutions; some adding B eucain or stovain, and others thinking this unnecessary. Solutions of the salts of magnesium and other salts of sodium give results as good as, but no better than, salt solutions. The following will serve every purpose: Stovain, gm. 0.1 ($1\frac{1}{2}$ grains), normal salt solution, 100 c. c. (3 1/3 fluid ounces); sterilize by boiling for half an hour, and then add ten to twenty minims of a 1 to 1,000 sterile solution of adrenalin.

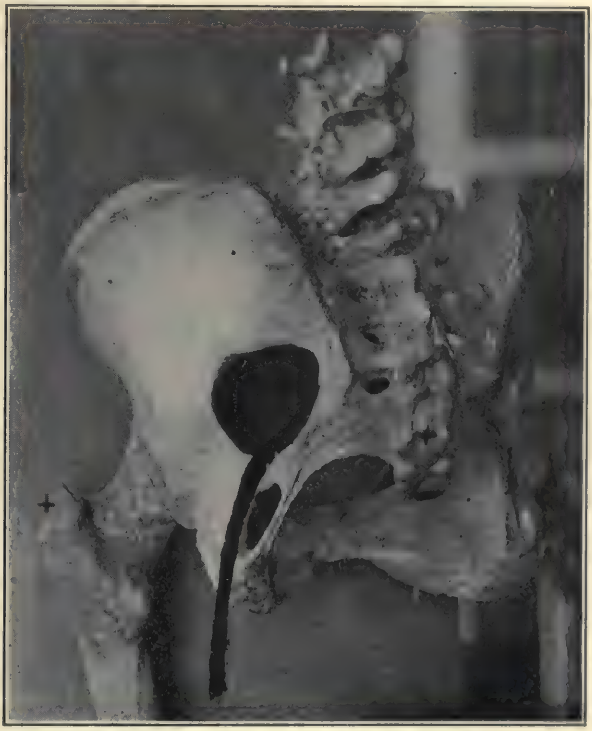

FIG. 1.—DRIED PELVIS WITH THE COURSE OF THE SCIATIC NERVE INDICATED IN BLACK. The crosses mark the sacrococcygeal junction and the upper end of the postero-external border of the great trochanter. The nerve is to be injected where it crosses the spine of the ischium just below its exit from the great sacrosciatic foramen.

PLACE TO INJECT.—If the pain is mainly in the peroneal distribution below the knee, injection into the peroneal branch, where it turns around the head of the fibula, may be sufficient; or, if a previous injection at the higher point has left some peroneal pain, a supplementary injection here is desirable. It is very easily done, unless the patient is too stout for the nerve to be felt. The fact that the needle has struck the nerve will always be indicated by a characteristic shooting pain. The best place to reach the main trunk of the nerve is where it passes over the spine of the ischium, immediately after leaving the pelvis through the great sacrosciatic foramen. This has the advantage of being the highest accessible point on the trunk, hence the injection is more likely to be above the seat of irritation. As the nerve here passes between a bony point and the pyriform muscle, it is itself one of the points likely to be irritated. It can be accurately located by means of anatomical landmarks, and the bone beneath the nerve gives definite information when the needle has penetrated far enough. To find the point on the surface which is perpendicularly over the spine of the ischium, measure from the middle of the sacrococcygeal junction to the upper end of the posteroexternal border of the great trochanter. Mark the junction of the inner and middle thirds of this line and then go one inch further out. This is the point where the needle is to be inserted, and it should be marked so that it can be found

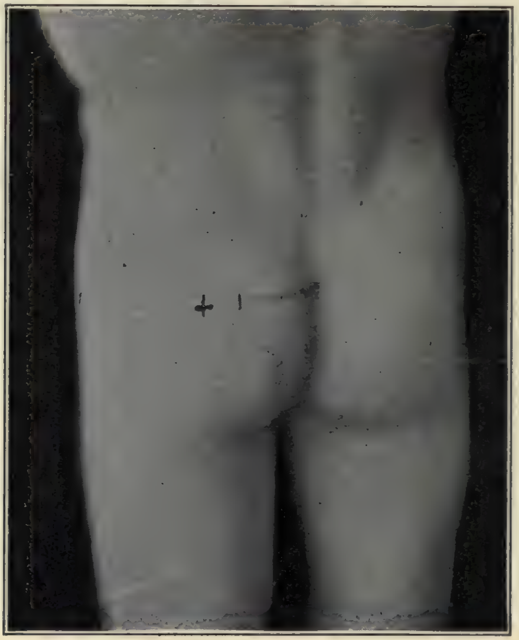

Fig. 2.—The cross marks the point on the buttock which is perpendicularly over the sciatic nerve where it crosses the spine of the ischium. This point is on a line joining the sacrococcygeal junction and the upper end of the postero-external border of the great trochanter, one inch external to the junction of the inner third of this line with its outer two thirds.

after sterilizing the skin. It also lies on a line joining the posterior-superior spine of the ilium, and the tuberosity of the ischium at the junction of its middle and inferior thirds, but this is not so good a guide because the tuberosity is hard to define. The landmarks given have been worked out especially by Richet and by Lévy and Baudouin (22), and have been thoroughly tested, clinically and on numerous cadavers, by Hecht (19).

Technique.—The buttock should be sterilized with alcohol and ether, and it is well to paint the point of insertion with iodin. This point and the tissue immediately beneath should be anesthetized with a little solution in a hypodermic syringe with a fine needle. This refinement is not strictly necessary, but patients with sciatica have already had all the suffering that is good for them, and even the most hardened will appreciate being spared whatever is unnecessary.

The needle must be a strong one, so as not to be broken by muscular contraction; 1.5 mm. (1/16 inch) in diameter, and at least 10 cm. (4 inches) long. It is an advantage to have it marked in centimeters. It should be beveled but blunt, and furnished with a stylet. The syringe should preferably be of metal, hold 100 c. c., and be readily attachable to the needle. Both must be sterilized by boiling in soda solution or in some other efficient way. Most operators recommend the lateral position, with knee and thigh semiflexed, or the knee-chest position, but Hecht prefers to have the patient lie on the abdomen with thighs and knees extended. This makes it easier to control disturbing motions.

The needle with syringe attached is inserted slowly perpendicular to the surface. As its point advances beyond the area already anesthetized a few drops of the solution are injected. When the nerve is reached at a depth varying between 4 and 10 cm. (1 3/5 and 4 inches) the patient always experiences a characteristic shooting pain in the heel or along the course of the nerve, usually with jerking of the muscles. It is essential to elicit this "control pain." The solution is now slowly and steadily injected and the needle withdrawn. The puncture is sealed with collodion and cotton. The patient is to remain in bed until the second day following.

Results.—In a large proportion of cases there is immediate relief of sciatic pain. If the injection is large, slight fever is apt to follow, and does not mean an accidental infection. A moderate degree of local pain and tenderness is to be expected. It can be treated by warm fomentations. In approximately two-thirds of the cases treated by this method cures have been obtained. Sometimes a single injection has been sufficient, but in the more obstinate cases two to five have been required. The method is as nearly safe as any direct action on a large nerve can be. A very few injections have been followed by peroneal paralysis, not nearly as many as one might expect, and such cases will probably recover completely, as the solution has no harmful chemical effect. For further details the reader is referred to the admirable article of Hecht, already cited, and to Lévy and Baudouin.

Surgical Operations.—In the past a small proportion of the obstinate cases of sciatica have been treated by exposing the nerve in the thigh and stretching it with considerable force so as to free it from adhesions and cause an appreciable elongation. The results varied greatly. Some cases were improved and ultimately cured, others were made worse. The general result was not nearly so good as is now obtained from injections; consequently, this operation should not be done until the injections have been thoroughly tried. Stretching the nerve without incision, but by extending the knee and then forcibly flexing the thigh on the trunk to a right angle or beyond, the so-called bloodless stretching, was also occasionally successful, but often made matters worse. It has properly fallen into disuse.

Pain not otherwise relieved will require analgesics or narcotics, as discussed in detail under the treatment of neuritis in general. In the incurable cases and those with the severest pain morphia hypodermically is indispensable, especially at night, but should, of course, be kept at a minimum and dispensed with as soon as possible.

REFERENCES

Since Chapters II, III, and IV are so closely related, the references for all three chapters have been combined in a single list at the end of Chapter IV, p. 360.

CHAPTER III

DISEASES OF THE CRANIAL NERVES

Howell T. Pershing

DISEASES OF THE OPTIC NERVE

Optic Neuritis

In any case of optic neuritis rest of the inflamed nerve is the first indication. This cannot be complete, for confinement in a dark room would be too depressing, but all use of the eyes in reading or work should be given up and bright light should be avoided. Wind, dust, and smoke are harmful. Care should be taken to see that the mind, as far as may be possible, is cheerfully occupied. Aside from rest the treatment of optic neuritis is almost exclusively that of its underlying cause.

From Organic Intracranial Disease.—Intense optic neuritis, known as choked disk, is marked by great swelling of the papilla, so that it projects forward into the vitreous and spreads out laterally, and by engorgement and tortuosity of the veins, perhaps with hemorrhages. Such a neuritis is generally caused by intracranial tumor. If the tumor is possibly syphilitic vigorous treatment with mercury, by inunctions or intramuscular injection, and iodid of potassium, should be begun immediately. Salvarsan is contraindicated. If this fails, or it is certain that the growth is not syphilitic, surgical intervention is the only means that will prevent death or blindness in the near future. The growth should, of course, be removed, if possible, but if, on account of its size, situation, or nature, this is impracticable, a prompt operation for decompression alone may be advisable, in order to save vision and to prolong, although not ultimately to save, life. Other organic intracranial diseases causing optic neuritis, usually not so intense as that of tumor, are: abscess, meningitis, disseminated sclerosis, and cerebritis. Their treatment is described elsewhere in this work.

In Toxic Conditions.—Many toxic conditions must be considered as possible causes demanding treatment. The most important of these are: syphilis, alcoholism, uremia, lead poisoning, arsenical poisoning, tobacco poisoning, pernicious anemia, leukocythemia, and obscure toxic conditions following acute infectious diseases, such as diphtheria, typhoid fever, and influenza. In the cases of metallic poisoning potassium iodid facilitates elimination, but, unless given in very small doses, cautiously increased, it may aggravate the condition.

From Suppuration of the Middle Ear.—It is not sufficiently known that suppuration of the middle ear may cause double optic neuritis, more marked on the side of the ear disease, without any apparent intracranial lesion. The connection is not understood, but it is probable that there is a continuous line of infection. In such cases the erroneous diagnosis of abscess may be made and a useless operation done; or the cause may not be suspected and nothing be done. The one thing essential is a radical operation on the ear and its adjacent bony cavities to remove all infected tissue. If this is successfully done the effect on the optic nerves may be surprisingly good.

In an extreme case of this kind in which neuritis and hemorrhages had at one time reduced vision to light perception, and in which the most exhaustive study failed to show the probability of any cause except suppuration of the ear, I saw improvement begin immediately after operation on the ear, and go rapidly to restoration of excellent vision for reading and writing. In such a case I would now use an autogenous vaccine, but would not delay operation. (Pershing, 33.)

From Myelitis.—The nature of the optic neuritis seen in occasional cases of inflammation of the upper part of the spinal cord is not known. Unfortunately, the primary condition is scarcely amenable to treatment of any kind, and tends rapidly toward a fatal issue, so there is no efficient treatment of the neuritis.

Cases Without Apparent Cause.—There are some cases of optic neuritis in which the most careful, extensive, and repeated examinations fail to indicate any cause whatever as even remotely probable. In these cases the immediate danger of blindness, the fear of fatal intracranial disease which cannot yet be recognized, and the inability to promise any substantial benefit from treatment make the physician feel most keenly the limitations of his art. Treatment, nevertheless, must be prompt and vigorous. After securing the necessary rest and tranquillity already mentioned, free elimination through the skin, kidneys, and bowels is to be secured. This can best be done by means of aspirin, hot packs, wet or dry, and saline laxatives. Tonics, especially strychnia, and perhaps iron, are indicated. Finally, even in cases that are certainly not syphilitic, mercury by inunction or injection and potassium iodid ought to be given to promote rapid absorption of the exudate in the nerve. As long as no irremediable cause appears, the case is hopeful.

I have seen perfect recovery in two cases of this kind. In one the neuritis was like that of intracranial tumor (choked disk), and vision was reduced to light perception, first in one eye and then in the other. In neither case did anything in the history before or after recovery give any clew to a possible cause. (33).

OPTIC ATROPHY

Atrophy of the optic nerves is either secondary to optic neuritis (including retrobulbar neuritis and neuroretinitis) or it is primary. The treatment of the secondary form must naturally be that of the preceding neuritis, for all cases of neuritis tend toward atrophy. Secondary atrophy may appear to be primary, although the ophthalmoscopic appearances are usually distinctive, because the preceding neuritis may have passed unobserved or may be retrobulbar. Moreover, the intracranial causes of neuritis may in rare cases cause atrophy without preceding inflammation. In any case of atrophy, therefore, all the possible causes of neuritis, intracranial, toxic, and infectious, should be systematically considered with a view to treatment. As in cases of neuritis, the possibility of syphilis being the cause should always be borne in mind, and, even if the case is not syphilitic, mercury and potassium iodid are indicated as long as there is any inflammatory exudate to be absorbed.

Primary atrophy is often part of a disease that is itself incurable, as tabes, paretic dementia, Friedreich's ataxia, muscular atrophy, or disseminated sclerosis. In almost all of these cases, even though considerable vision remains when treatment is begun, the destruction of the remaining fibers goes on uninfluenced. Nevertheless, there are enough favorable exceptions to make treatment worth while in every case, and, in disseminated sclerosis, while the disease is incurable, any particular part of it may undergo arrest or even notable improvement. In cases of tabes with optic atrophy antisyphilitic treatment must be moderate and cautious. As in optic neuritis, salvarsan is contraindicated. The possibility of lead or arsenic being the cause of atrophy, primary or secondary, must never be forgotten, and the search for the lead line on the gums, the arsenical pigment spots on the skin, and either metal in the urine must be thorough, as well as the study of the patient's history and occupation.

After any possible cause has been treated as effectually as may be possible, the atrophic process may to a certain extent be combated by strychnia. Oculists commonly give strychnia once daily hypodermatically in the temple, gradually increasing the dose up to gm. 0.015 or 0.020 (¼ to 1/3 of a grain). I think it wise to follow this practice, but would expect the same results from injections into any other part of the body or from administration by the mouth.

The galvanic current can be applied by putting a pad of moist cotton over the closed lids and applying the cathode to this while the anode is on the back of the neck. The current is to be turned on and off gradually, and to be allowed to flow steadily at a strength of from three to six milliampères. I have never seen this do any good, but Schmidt-Rimpler (39), with his great experience, is sure that it does. Of late years the high frequency current has been highly recommended. I have had no experience with it.

NEURITIS OF THE THIRD, FOURTH, AND SIXTH NERVES

Paralysis in the domain of these nerves is a common and very important symptom in intracranial tumor, meningitis, thrombosis of the cavernous sinus, tabes, bulbar paralysis, spinal muscular atrophy, myasthenia gravis, and disseminated sclerosis. In any of these diseases other symptoms will probably be so combined with the ocular paralysis as to lead to the more general diagnosis and the appropriate treatment. Various infections may cause paralysis of these nerves, by far the most important being syphilis, which is relatively common and usually yields promptly to treatment, unless too long delayed. Of the acute infections, diphtheria, influenza, scarlatina, measles, and typhoid fever occasionally cause ocular paralysis either through a meningitis, which is almost invariably fatal, or by a more localized action, whose prognosis is far more favorable.

Forms of intoxication which may cause ocular paralysis are alcoholism, diabetes, uremia, ptomain poisoning, and occasionally plumbism. These nerves are especially sensitive to such drugs as the belladonna and cocain groups and aconite. Transient paralysis in this field has followed spinal anesthesia by cocain and stovain. It has also appeared as an untoward result, usually transient, of the deep injection of alcohol into the middle branch of the trigeminus for neuralgia. It is one of the risks of operation at the base of the skull, especially that for extirpation of the Gasserian ganglion. Finally, from the absence of other probable causes and a history of exposure, some cases must be attributed to cold and regarded as analogous to the ordinary form of facial paralysis; in fact, both forms, ocular and facial paralysis, sometimes appear together on the same side after exposure to cold.

Aside from the causal indications suggested by the foregoing, all acute cases and the infective and toxic ones should be treated by rest in bed, sweating, diuresis, and such purgation as is well borne. Counterirritation by small blisters on the temple and local blood-letting by natural or artificial leeches are valuable in the more inflammatory and congestive cases.

Electricity cannot readily be applied to the muscles of the eyeball, and Oppenheim warns against trying to reach them with fine electrodes. The galvanic current may, however, be applied by placing a pad of warm moist cotton over the closed lids, the cathode on this, and the anode on the back of the neck. Two to five milliampères may be used, or just enough current to cause contraction when the cathode is applied to the facial muscles and the current closed. The current should flow steadily when applied to the eye, as the shock of making and breaking it would be too irritating. When the acute symptoms have subsided strychnia in full doses is advisable as a nerve tonic.

The annoying diplopia may be obviated with advantage for a time by the use of a light bandage or opaque glass over the affected eye. The use of the eye, especially such as calls for action of the weakened muscles, must only gradually be resumed, taking care to avoid excessive fatigue. Binocular vision may sometimes be restored by prescribing prisms, but they cannot satisfactorily correct any high degree of insufficiency. Operation on the muscles may be advisable, but only after other means of restoring balance have been exhausted, and there is reason to believe that the degree of defect is fairly constant and not too great. This is a special question for the oculist in each case.

Periodic Ocular Paralysis.—Cases of this rare affection probably have different causes. Some are clearly associated with migraine, constituting the *migraine ophthalmoplégique* of French writers. These should be treated like other cases of this neurosis, with extraordinary care to avoid ocular fatigue. They have a comparatively good prognosis, but not so good as cases of ordinary migraine. In other cases, even though some of them seem clearly to be migrainous at first, the periodic attacks are early symptoms of organic intracranial disease, either vascular or degenerative. Hence, the prognosis must be guarded and the treatment must be according to the causal indications furnished by the history and physical examination.

TRIGEMINAL NEURITIS

Neuralgia, which is by far the most frequent disease affecting the trigeminal nerve, is not regarded as an organic disease, and is treated under the neuralgias. Herpes zoster is also separately considered.

Almost all other diseases of this nerve are secondary to tumor, inflammation, aneurysm, or injury at the base of the skull, and these must be treated according to their nature and seat. Among them syphilis is especially frequent, and in doubtful cases thorough antisyphilitic treatment should be given. The intense pain caused by organic disease affecting this nerve must be treated by analgesics, and, these failing, by injections, as in cases of neuralgia. Even when the irritation is central to the injection shielding the diseased area from impulses starting in the periphery may do considerable good.

Primary neuritis is exceedingly rare; when it occurs it is generally in association with neuritis of the seventh nerve or a part of multiple neuritis. The treatment is that already indicated for pain in addition to the treatment of the principal disease. Paralysis of the muscles of mastication is to be treated with the galvanic current, as in other cases of neuritic paralysis.

NEURITIS OF THE SEVENTH NERVE

(*Facial Paralysis—Bell's Palsy*)

The great majority of cases of facial paralysis are of the so-called rheumatic type, that is, they are due to exposure of the face to cold or a prolonged draft of air, or the cause is not ascertained. Nevertheless, the numerous other possible causes of the rarer cases are first to be considered. In all of these treatment appropriate to the original cause is to be carried out, as far as may be possible, to be followed later by the local treatment advised for the rheumatic type.

Intracranial Disease.—Intracranial new growths, inflammations, and injuries to the base of the skull may affect this nerve before its entrance into the internal auditory canal. In such cases other nerves are almost necessarily involved, especially the eighth, and the general signs of intracranial disease will be present. Among the causes of such disease syphilis is of the greatest importance.

From Disease of the Temporal Bone.—The long curved course of the nerve through a narrow bony canal, from the internal auditory meatus to the stylomastoid foramen, exposes it to attack from any affection of the bone, and at the same time makes any swelling in this part of its course an immediate cause of damage by pressure. Fracture, caries, tuberculosis, or gumma of the bone, with hemorrhage, inflammation, or pressure in the canal, may easily cause paralysis, which usually comes on so rapidly as to appear sudden.

Suppurative disease of the ear is the most important cause of disease of the temporal bone. The mere coincidence of facial paralysis with disease of the ear does not, however, without further investigation, warrant the assumption that the paralysis is caused by the disease of the ear. The two conditions may possibly be entirely independent, or they may both be dependent on a common cause, such as exposure or injury. Operations on the diseased ear not very rarely cause facial paralysis, because of the very close relation of the nerve to the aural cavities and especially to the antrotympanic passage.

When suppurative otitis causes facial paralysis it is almost always chronic and has caused necrosis of the bone. Early treatment by a skilled otologist would prevent this stage being reached, but when the nerve is involved the prospect of its restoration is not good. In any case, however, the most thorough treatment of the ear and its adjacent bony cavities is urgently demanded, irrespective of the effect on the nerve, but with the hope that it will recover if intracranial infection be prevented and life be saved. Pus must be evacuated, all necrotic and granulomatous tissue removed, and drainage maintained. According to the condition of the ear and temporal bone, any operation may be necessary, from paracentesis to the radical cleansing of the mastoid, antrum, and tympanum. In some cases it may be advisable to remove enough of the facial canal to free the nerve from pressure and infected surroundings, although this is difficult and, of course, involves a fresh danger.

In traumatic cases, including those caused by operation, primary suture should be done if possible.

Certain specific diseases, including influenza, typhoid fever, diphtheria, mumps, erysipelas, and tonsillitis, occasionally cause facial paralysis, no doubt by an extension of the adjacent local infection to the nerve near its exit from the stylomastoid foramen.

Rheumatic and Idiopathic Cases.—The rheumatic cases if seen early should have leeches applied below the ear or a blister over the mastoid process, in the hope of reducing the assumed congestion and swelling in the facial canal. Revulsion by a hot mustard foot bath, followed by sweating, diuresis, and purgation, is appropriate. Aspirin has the advantage in such cases of being a salicylate, favoring the prompt elimination of wastes of the uric acid class, and also of being a powerful diaphoretic.

If there is pain limited to the external auditory meatus and inner surface of the auricle, it is explained by Ramsay Hunt's theory that the facial is not a purely motor, but a mixed, nerve having sensory fibers whose distribution is in the meatus and auricle, whose root ganglion is the ganglion geniculatum, and whose sensory root is the nerve of Wrisberg or portio intermedia. If pain extends beyond this area it means that some branches of the trigeminus are also involved. In either case the pain is best treated by warm poultices behind and in front of the ear or by the dry heat of a water bag, which will usually give relief.

Cold applications should always be avoided, and in the course of treatment special care is to be taken to avoid chilling the face. One of Oppenheim's cases of facial paralysis was caused by the application of ice to the neck as a remedy for tonsillitis, and it is a familiar fact that, long after a nearly perfect recovery, exposure of the face to cold causes renewed stiffness and weakness. The inability to close the eye is often a source of much annoyance and also of some danger, as the tears are not properly distributed and the exposed cornea is injured by drying, in addition to the irritation of the whole eye by light and dust. The patient should be instructed to close the eye frequently by gently pressing the lids together with his fingers and thumb, thus sheltering and moistening the eyeball from time to time. At night the lids may be held closed by a wad of moist cotton, held in place by a light bandage. This often adds greatly to the patient's comfort and may also be used during the day.

The sound side of the face should frequently be drawn over toward the median line and the muscles of the paralyzed side drawn away from it. From the beginning light, skillful massage may also be of benefit, but the physician should be sure that it is really light and not overdone. If electrical treatment is properly carried out, professional massage is rarely necessary.

Electricity for treatment is contraindicated during the first week or ten days, and its use for diagnosis is not so valuable as later. There is no objection, however, to faradic or galvanic tests at any time.

After ten days the facial muscles should be carefully tested with both the faradic and galvanic currents for the purpose of prognosis. Complete loss of faradic irritability and greater anodal closure contraction than cathodal closure contraction, both being sluggish (this combination of reactions constituting the reaction of degeneration), mean that the case is severe and that no marked improvement will be manifest until after three months. On the other hand, if cathodal closure contraction is greater than anodal closure contraction and both are quick, but faradic irritability is lost, the case is only moderately severe and earlier improvement may be expected. If faradic irritability is retained the case is one of the lightest and may recover in from two to four weeks. Most of these light cases would, no doubt, recover without treatment, but they do better with it. Both the severe and the light cases should receive galvanic but not faradic treatment.

To act on the lesion itself a small electrode or the well-padded curved edge of a larger one should be gently applied between the jaw and the mastoid process; this should be the cathode and the anode may be over the cheek. The current is turned on gradually, allowed to flow at a strength of three to six milliampères for three to five minutes, and gradually turned off.

To maintain and improve the condition of the paralyzed muscles the indifferent electrode is applied to the back of the neck, and the active one, usually somewhat smaller, is applied to the orbicularis oris; then the current is gradually increased and slowly interrupted until closing it produces a moderate contraction. This will occur first with the cathode on the muscle in mild cases, but with the anode in severe ones. Whichever arrangement of poles produces the greater contraction with the same current is to be chosen. The active electrode is now applied to the different muscles in turn, the current being closed a few times to secure contractions of each muscle. It is better to leave a muscle and come back to it several times than to fatigue it by causing too many contractions at once. The strength of current will vary from one to six milliampères, according to the area covered by the active electrode and the muscle acted upon. The essential thing is to get moderate contractions without pain or fright. About the eye and on the forehead the susceptibility to pain is greater and weaker currents must be used. Special care must be taken in treating the orbicularis palpebrarum not to irritate the eyeball. The upper lid is drawn down gently over the eye before the electrode is applied. In the later stages, in addition to slow interruptions of the current, the muscles about the mouth and cheek, but not those near the eye, may be stroked with the active electrode, so as to give a kind of electric massage which is very stimulating. The whole treatment should last from ten to fifteen minutes and be repeated from three times weekly to daily, according to the circumstances.

The general health of the patient, both physical and mental, is to be guarded and built up by all available means. Of medicines, aside from those that fulfill a causal indication, like mercury, or are called for by incidental disorders, strychnia is the most important on account of its general tonic properties and its special effect on motor neurons. Food, moderate exercise, and fresh air are important, but the face must be carefully guarded from cold and wind even after convalescence is well advanced. Although paralysis of the face, even of the cerebral type, is practically never seen as a part of hysteria, there is no doubt that emotional conditions can in some way affect peripheral facial palsy. Oppenheim saw it twice in the same patient, each time apparently caused by fright. We should not neglect, therefore, to foster courage and tranquillity.

Many severe cases make only a partial recovery. Some power of voluntary contraction comes back, but along with it comes a tonic contracture of the muscles, so that the nasolabial and other furrows become deeper than on the sound side, which, being smoother, may now be mistaken for the paralyzed one. On voluntary or emotional action, however, the mistake is quickly corrected as the appearance is reversed, the contractions on the sound side being of much greater amplitude. There is no remedy for this state. When it appears electrical applications should be discontinued, for they can do little or no good; but, on the other hand, they should not be regarded as having helped to cause the contracture. Balls have been carried in the mouth to distend the cheek and massage with stretching movements has been tried. There is no objection to such stretching, but it accomplishes so little as to be hardly worth while.

Late Operation.—In traumatic cases where no considerable degree of recovery has taken place during a year's treatment surgical intervention may still give hope of improvement or even recovery. If the central part of the nerve is sound, freeing its trunk from the pressure of exudate or scar tissue (neurolysis) may permit recovery to begin. Or the damaged part of the trunk may be excised and the ends brought together and sutured; this, however, is rarely possible. Union of the peripheral part of the facial with the central part of the hypoglossal or spinal accessory has been successfully accomplished. Return of the muscular tone, so that the appearance of the face in repose has been improved, and some degree of voluntary motion of the paralyzed side, have been restored by the successful operations of this kind. The voluntary motions, however, are crude, the patient having to think of moving the shoulder or tongue, as the case may be, and are marred by undesirable associated movements of these parts. In emotional expression the defect is as glaring as ever (Lannois and Porot, 21). I have seen one remarkably successful case of this kind, in which W. W. Grant of Denver (14) sutured the peripheral part of the facial to the central part of the spinal accessory, and at the same time, to remedy the paralysis of the trapezius and sternomastoid muscles caused by section of the spinal accessory, cut the descendens hypoglossi and sutured its central part to the peripheral part of the spinal accessory. There was very little inconvenience from weakness of the trapezius at any time. Fifteen weeks after operation slight movement of the facial muscles appeared in association with efforts to raise the shoulder. The restoration of the facial muscles in repose was eventually nearly complete and strong voluntary contractions were possible. The annoying association of movements became less marked and the shoulder movements were normal or nearly so. The disparity of the two sides of the face in emotional expression remained.

Congenital Facial Paralysis.—Congenital cases, if due to defective development, are not amenable to treatment of any kind. If caused at birth by pressure of forceps or by the manipulations of a breech delivery, they should be treated in the way already described with galvanic electricity. The result is usually good.

DISEASES OF THE EIGHTH NERVE

(*Nervous Deafness, Aural Vertigo*)

The eighth nerve is really two nerves, the auditory or cochlear and the vestibular nerve. The vestibular nerve has nothing to do with hearing. It carries sensory impulses starting in its nerve endings in the ampullæ of the semicircular canals and in the utricle. The inertia of the endolymph causes a varying pressure on these different nerve endings, corresponding to the different changes in the position of the head and to the motions of the body as a whole, thus arousing impulses for the special sense of equilibrium and bodily motion. As the two nerves, while distinct, are in the same sheath, and as the labyrinth is a continuous structure, disease affecting the one is practically certain to affect the other.

Accordingly two sets of symptoms occur together, as an indication of disease of these nerves or of their special endings in the labyrinth. Irritation of the cochlear nerve causes various subjective noises, while irritation of the vestibular nerve causes vertigo, which, when intense, is accompanied by vomiting, as in seasickness and in Ménière's disease. But, if the morbid process continues, what was at first mere irritation with irregular increase of function becomes destruction with loss of function. Accordingly, deafness is soon added to the subjective sounds, and when it has become absolute they generally cease. In the same way absence of any sense of motion gradually replaces the false sense of motion which ultimately entirely disappears. It has been noted of certain persons with total deafness that they are never seasick and cannot be made dizzy by being whirled in a revolving chair.

The treatment of the auditory symptoms of disease of the eighth nerve is only rarely successful even when begun early. Tinnitus is notoriously obstinate and the deafness is still more so. Patients generally come to the otologist when it is far too late for him to accomplish anything. Vertigo, on the other hand, often appears earlier than the tinnitus or deafness, and, being a symptom of irritation, of impending rather than actual destruction, it is, like pain, a symptom that can be alleviated and a valuable warning that something must be done. Moreover, it commonly brings the patient to the neurologist or internist instead of to the otologist, and that is why the subject is treated here, instead of being left to works on diseases of the ear.

If the cause is intracranial, a tumor, abscess, aneurysm, or meningitis, other nerves, especially the seventh, will probably be involved, and the general signs of intracranial disease will be present, so as to make its location, and perhaps its nature, clear. An intracranial infection, especially syphilis and epidemic meningitis, may extend outward along the nerve and attack the labyrinth, usually on both sides at the same time or in rapid succession. Hence, these two diseases, along with scarlatina, which attacks the labyrinth from the opposite direction, through the throat and middle ear, are the prolific sources of deafness and deaf mutism.

The treatment of the cases starting within the cranium must be that of the primary disease, which is rarely successful, and even when successful in other respects very seldom restores hearing. In epidemic meningitis, however, the outlook, at least for prevention of disease of the labyrinth, is much better since the introduction of the antimeningitic serum. Lumbar puncture should be done early, and, if the cerebrospinal fluid is turbid, the serum should be injected immediately. In syphilitic cases the largest doses of mercury and potassium iodid that the patient will tolerate should be used as early as possible. If hearing is already lost, it will be regained in only a small proportion of cases. Chavanne (5) advises that injections of pilocarpin, as described below, be added to the antisyphilitic treatment, so that, in the event of failure to restore what is lost, or even to save what is left of hearing, the physician can feel that the most intensive treatment has been employed. I know of no experiences with salvarsan in such cases, but should regard it as contraindicated, as in optic neuritis, on account of its tendency temporarily to increase congestion and swelling in an active syphilitic lesion. More frequent than the intracranial involvement of the eighth nerve are the cases in which the labyrinth is attacked from the peripheral side through the middle ear or temporal bone. In all such cases the most skillful and thorough and the earliest possible treatment by an otologist is imperative. In diseases like scarlatina, influenza, typhoid fever, and mumps most cases of serious disease of the ear could be prevented by careful attention to the nose and throat, repeated examination of the ears, and immediate treatment of otitis as soon as detected.

If there is no intracranial disease and no disease or injury of the middle ear or temporal bone, the symptoms are caused by disease within the labyrinth.

If the onset is sudden or acute we have to deal with hemorrhage, congestion, serous exudation, or inflammation. Here, again, we must think of syphilis as a possible cause of any of these pathologic conditions, for it attacks the labyrinth primarily as well as by extension. In case of doubt the treatment should be prompt and vigorous, as mercury and iodid have a favorable tendency even in non-syphilitic cases. Gouty and rheumatic conditions, although generally chronic in themselves, often cause labyrinthine symptoms of acute or sudden onset. In any acute case perfect rest in bed in a quiet room is the first essential. Even turning in bed must be avoided or done slowly and cautiously. The first attack is often precipitated by a sudden turn. Derivation to the feet with a hot mustard foot bath may be employed at once, but the patient should not sit up. The bath can be brought to the side of the bed, and the patient should lie still, while the nurse immerses the feet. Free purgation with calomel and salines is desirable. Pilocarpin is not to be used in this stage. Even in gouty cases salicylates are to be avoided on account of their tendency to irritate the labyrinth. Later they may be used with caution. Colchicum may be given if otherwise indicated, because its tendency is toward intestinal irritation, which will do no harm. Blood-letting or blistering over the mastoid undoubtedly tends to relieve the internal congestion and should be employed. To relieve the intense distress, acetphenetidin and bromids are serviceable. If vomiting prevents their retention bromid may be given by rectum.

When the acute symptoms have subsided, if deafness and vertigo still remain, pilocarpin should be used hypodermically. This drug causes leukocytosis with free sweating, salivation, and diuresis, together with more or less nausea and prostration. The object is to secure the maximum absorption of exudate with the minimum of undesirable symptoms. The dose is gm. 0.01 to 0.02 (1/6 to 1/3 of a grain) for an adult, but the first dose should be only gm. 0.005 (1/12 of a grain) to test the patient's susceptibility. Children should receive the proportionate dose according to the usual rule. The dose should be increased until it causes free sweating, with salivation and perhaps some nausea. The time to choose is a few hours after a light meal. The patient must be in bed wrapped in blankets, and, after the sweating, must remain in bed for some hours at least, and be kept warm and dry. These injections are to be repeated every day or every other day according to the tolerance of the patient. If after twelve injections there is no improvement in hearing, the treatment should be given up. The deafness is incurable, although the vertigo may be greatly improved or have disappeared altogether (Chavanne, 5). If there is some improvement the injections should be resumed, after a few days' interruption, and as many as thirty may be given altogether, after which no further good may be expected from it. If the pilocarpin should cause alarming symptoms, such as pain, diarrhea, vomiting, or dimness of vision, a hypodermic injection of strychnia, 0.002 gm. (1/30 of a grain), and atropia, 0.0007 gm. (1/100 of a grain), is the best corrective.

Chronic disease of the labyrinth is essentially incurable as far as the deafness is concerned, no doubt because the neurons of the cochlear nerve, having their bodies in the spiral ganglion, are partially or totally destroyed. The sensory loss is analogous to that caused by destruction of neurons in the retina or in the posterior root ganglia. The further progress of the disease, however, and the liability to attacks of the Ménière type, should be combated by constitutional and symptomatic treatment. The gouty diathesis and arteriosclerosis are the constitutional conditions most commonly present. Free elimination by means of moderate doses of calomel and saline laxatives does much good. Salicylates, if otherwise indicated, may be used, but only in moderate doses and with extra caution. Potassium iodid may often be given with advantage, as it probably has some good effect on arteriosclerosis and favors absorption where that is possible.

The treatment of tinnitus and Ménière's attacks with massive doses of quinin, introduced by Charcot a generation ago, seems odd on first consideration, because such doses are well known to endanger even the healthy labyrinth. Charcot was led to try this by observing that, when deafness became total, the tinnitus and vertigo generally disappeared, so in his first cases he deliberately chose to sacrifice what little hearing might remain in order to relieve the other symptoms. This, of course, should be taken into account and the patient should understand its probable effects. Charcot gave gm. 0.5 to 1.0 (7½ to 15 grains) daily for several weeks. His report of improvement in many cases was confirmed by others, and there is no doubt that this treatment may be advisable in certain cases, but it is far from being generally applicable (Frankl-Hochwart, 9). The best medicine to relieve tinnitus and vertigo is some form of bromid or hydrobromic acid.

I prefer sodium or strontium bromid, gm. 2.0 to 4.0 (30 to 60 grains), daily, the doses being apportioned so as to get their maximum effect when relief is most needed.

Galvanic electricity applied to the ear has been tried. While it undoubtedly can influence the labyrinth, as shown by the vertigo excited by moderate currents and the subjective sounds by very large ones, there is no satisfactory proof that it does good.

Thyroid extract has also been tried without substantial success (Chavanne, 5).

Babinski has recommended simple lumbar puncture, and has found it to have a remarkably favorable effect, especially on the vertigo, rarely on the deafness. Its good effect on the vertigo has been confirmed by J. J. Putnam (36). Some of his patients had complete relief for many months after a single puncture, and relapses were relieved by subsequent punctures.

The general health of the patient must be carefully guarded and built up by every possible means. The mental condition is often one of great anxiety or depression, and this should be treated as carefully as the physical symptoms.

NEURITIS OF THE NINTH NERVE

(*Glossopharyngeal Paralysis*)

Disease of the ninth nerve practically never occurs alone, but always with lesions of other nerves, especially the tenth and eleventh. The involvement of this nerve is recognized by disturbance of taste in the posterior two-thirds of the tongue, and difficulty in swallowing. What is said in the following section of the intracranial, cranial, and constitutional causes of vagus neuritis applies equally to this nerve. The treatment and prospects of success depend on the particular cause.

The difficulty in swallowing makes the affection a very serious one, and calls for extreme care in feeding the patient. Semisolid foods, custards, or preparations of milk thickened with flour, rice, or other forms of carbohydrate food, made more nutritious by the addition of eggs, are more easily swallowed than either solids or liquids. When these cannot be taken in sufficient quantity, milk and eggs must be given by a nasal or esophageal tube. Rectal feeding may be used to supplement this, but it is not sufficient alone. Thirst may well be relieved by enemata of normal salt solution, especially by the drop method.

Bernhardt recommends electrical treatment of the pharyngeal muscles.

NEURITIS OF THE TENTH NERVE

(*Pneumogastric Paralysis*)

The vagus in its long course is subject to many possible causes of disease or injury. In its intracranial course from the medulla to the jugular foramen the principle cause of disease is syphilis, but it may also, with adjacent nerves, be damaged by hemorrhage, tumors, aneurysm of the vertebral artery, or caries of the temporal or occipital bone. As syphilis is the most common of these intracranial causes, and is also far more amenable to treatment than any of the others, every means must be taken to decide whether or not it is present. In doubt, even while waiting for the report of a Wassermann test, mercury and iodid should be given in full doses.

In the neck the nerve is in danger principally from disease of the lymphatic glands, usually tuberculous, sometimes malignant. It is here also occasionally damaged by pressure from an aneurysm of the carotid artery. It may be involved in wounds of all kinds, and is not so very rarely injured in operations.

I once counseled C. A. Powers in the case of a young man who recently, in another city, had been operated on for tuberculous glands of the neck. The operation was said to have been hastily concluded on account of hemorrhage, and immediately after recovery from the anesthesia the patient had most violent and distressing paroxysms of coughing and dyspnea. His attacks continued at short intervals and seemed dangerous, but nothing that we administered had any substantial effect. We suspected a ligature of the vagus, and this was soon confirmed by the appearance of the end of a heavy silk ligature in a fistulous opening. Following this, Powers easily reached the bight of the ligature, which was cut and removed. The paroxysms were greatly relieved at once, and soon ceased altogether. The diseased glands were afterwards successfully removed.

In cases in which the disease is caused by the pressure of tuberculous glands, surgical treatment may be supplemented by the use of tuberculin or the Röntgen ray, but, when nerves are involved, surgical removal of the glands is generally imperative.

In the chest the nerve may also be damaged by diseased glands, and it may be involved in pleuritis or pericardial exudate or in any disease of the mediastinum. The recurrent branch may be involved alone, and, as is well known, paralysis of the laryngeal muscles supplied by this branch is often the earliest recognized symptom of aneurysm of the aorta. Paralysis of the left recurrent laryngeal nerve may occur in mitral stenosis from the pressure of the dilated left auricle.

The vagus suffers, generally as a part of multiple neuritis, in various specific diseases, as diphtheria, influenza, typhoid fever, and pneumonia;

also in intoxications, as alcoholism, phosphorus poisoning, arsenical poisoning, and plumbism. The indications for treatment in these cases are given under multiple neuritis, and also under the various original diseases.

An expert laryngologist may, with specially devised instruments, apply galvanism internally to the weakened laryngeal muscles. Or, as Bernhardt specially recommends, the easier percutaneous method may be used. In this method a button-shaped electrode, the cathode, is applied between the inner border of the sternomastoid muscle and the trachea, just below the cricoid cartilage, and is gently pushed backward. The other electrode may be over the thyroid cartilage on the other side. The current should be mild—one to three milliampères. The faradic current can also be used, but in my opinion it ought not to be.

If suffocation is threatened by paralysis of the abductors of the vocal cords, either intubation or tracheotomy should be performed, preferably tracheotomy.

NEURITIS OF THE ELEVENTH NERVE

(*Spinal Accessory Paralysis*)

As the roots of this nerve come from the cervical cord the causes of its disease are to be sought first in the upper part of the spinal column. Pott's disease of the upper cervical vertebræ and tuberculous or syphilitic inflammation of the membranes of the cord are the conditions which may affect it here. In its course from the foramen magnum to the jugular foramen it may be affected by disease of the occipital or temporal bone or by meningitis, tumor, or aneurysm. Treatment is hopeful only when the spinal or intracranial cause is syphilis. In the neck it may be affected by disease of the lymphatic glands and by wounds and operations. In case of severance by wound or operation primary suture should be done, or, if this has not been done, suture or anastomosis with another nerve may be attempted later.

The resulting paralysis of the sternomastoid is not a very serious matter but that of the trapezius allows the scapula to sag downward and forward on the chest so that the head of the humerus may press on the ulnar nerve or on the brachial plexus generally, thus causing pain, deformity, and serious impairment of the functions of the arm. This calls for special orthopedic apparatus to raise the shoulder and hold it back. Gaupp has devised such an apparatus for bilateral paralysis of the trapezius. In his case it relieved pain and allowed the arm to be properly used.

As long as there is hope of recovery electricity should be used in the two ways described under the treatment of neuritis in general.

NEURITIS OF THE TWELFTH NERVE

(*Hypoglossus Paralysis*)

The nucleus of the hypoglossus is affected in a number of central nervous diseases involving the medulla, but these do not concern us here. Peripheral disease is correspondingly rare. In its short intracranial course the hypoglossus is, like the other nerves at the base, liable to be damaged in hemorrhage, tumor, or inflammation. Its close proximity makes it fairly sure to be involved in case the occipitoatlantoid joint is diseased. After leaving the skull it is still more rarely affected, but even in its sheltered situation it is occasionally reached by a bullet or stab wound or compressed by a gland or tumor.

Little effective treatment is possible, and many cases end fatally from the accompanying conditions, but the same principles apply as in the case of other cranial nerves. Syphilis, if taken in time, can be cured. Suturing the nerve if severed by a bullet or knife is not necessarily beyond surgical skill. Electricity may be applied with one electrode directly on the tongue and the other over the great horn of the hyoid bone.

MULTIPLE NEURITIS

Treatment of Causes

While in mononeuritis the principal causes are local, and constitutional predisposition is generally only incidental, in multiple neuritis the converse is true, the principal cause is always constitutional, and local aggravations, if they exist, are only incidental. Nevertheless the constitutional cause in multiple neuritis does not, as one might naturally expect, act equally on the nerves throughout the body. The peculiar symmetrical and peripheral distribution of the symptoms shows that there is a special predisposition in the neurons of the musculospiral and peroneal nerves and that this is greatest in those having the longest axis cylinders.

In any case of multiple neuritis the first step in treatment is toward the removal of the constitutional cause. This cause is usually a poison which may be introduced from without, as in alcoholic and arsenical neuritis, or be produced within the body, either through an error of metabolism, as in diabetic neuritis, or through the action of a living germ, as in diphtheritic paralysis.

The poisons from without the body that most frequently cause neuritis are alcohol, arsenic, and lead; those that may occasionally, but far more rarely, be encountered are ptomains, copper, mercury, carbon monoxid, carbon bisulphid, anilin and phosphorus.

Among the metabolic poisons that of diabetes is by far the most important. Gout is recognized as having considerable influence as a contributory cause, but Oppenheim doubts that any well marked case has been due to it alone, and he also considers uremia a doubtful cause. In a very small number of cases gastrointestinal disease or disease of the liver has been regarded as the cause. The cases caused by prolonged overexertion with exposure to cold, as in a swimming match, may be due to poison produced by changes in metabolism.

Of the infections diphtheria is clearly the most important, but it would be difficult to name an acute specific infection that may not cause multiple neuritis. Thus cases have apparently been caused by typhoid fever, influenza, scarlatina, puerperal fever, and other forms of septicemia, acute rheumatism, small-pox, whooping cough, erysipelas and malaria.

Of the chronic infections leprosy has multiple neuritis as its most important symptom group. In the later stages of tuberculosis it is common to have the diagnosis of multiple neuritis suggested by pains, but not confirmed in the subsequent course by other signs of nerve lesion. In a much smaller number of cases, where a mixed infection is probably the chief cause, there is actual neuritis, as shown by loss of tendon reflexes, and atrophic paralysis.

Syphilis, a common cause of multiple lesions of the cranial nerves, is a rare cause of multiple neuritis affecting the spinal nerves, so rare that Oppenheim says that the diagnosis should always be made with great reserve.

Beriberi cannot be definitely classified as an infection or intoxication, as the cause is not yet known, although it is symptomatically a form of multiple neuritis.

Finally, it is probable that mere defect of nutrition without infection or intoxication may cause multiple degeneration of the nerves, as in some cases of severe anemia and cachexia.

These causes have been mentioned at some length because in treatment it is necessary to keep in mind the possibility of one of the rarer or more obscure causes being an important factor. Even when the main cause is certainly known it may not be sufficient to treat it alone, as there may be a combination of two or more constitutional causes in the same patient. Thus alcoholism, with its well-known lowering of resistance to infection, may be combined with rheumatism, pneumonia, influenza, syphilis, tuberculosis, or any other infectious disease. Gout is generally added to lead poisoning and often to alcoholism, while intestinal autointoxication may be an important complication of any case.

Alcoholism.—In cases of neuritis due to alcoholism immediate and total abstinence from alcohol in all its forms should be the rule. This rule is often very difficult to enforce, not only in lighter cases where the patient is still able to go about, but also in cases of complete disability, unless the physician can control every person who has access to the patient. The tendency to deception, which is often most artistic, and the great danger of continuous surreptitious consumption of alcohol must constantly be borne in mind, most of all when the patient is a woman. In one of my fatal cases the patient, a seemingly frank and ingenuous woman, absolutely denied the use of alcohol in any form, then conscientiously corrected herself and told how some months before she had taken a single glass of sherry. I found that her daughter, aged nine, was going daily to a neighboring drug store and bringing back a pint of brandy, which the child naïvely said did her mother "lots of good."

Even in cases under complete control in a hospital it is wise to prescribe what will be regarded as a substitute and by quieting restlessness and irritability allay the intense craving for a stimulant, as in the following formula:

℞

Tr. opii deod.,	3	mxlv
Tr. capsici.,	6	ʒiss
Tr. nucis. vom.,	15	ʒiv
Flext. casc. arom.,	20	ʒv
Elix. calisayæ, q. s. ad.,	60	℥ii

M. S.—Teaspoonful in water every three hours.

Gowers, Bernhardt, and Oppenheim all say that in cases with a weak heart wine or brandy should for a time be prescribed in proper doses rather than abruptly to withdraw all forms of alcohol. In my own cases of alcoholism in its various forms I have never thought it necessary thus to "taper off," but, relying on strychnia and digitalis with the most perfect rest in bed to support the heart, have withdrawn alcohol at once. Only when the heart remained dangerously weak in spite of this treatment would I go back to brandy or wine.

Should delirium tremens be present as a complication it is to be treated as if it existed alone, with rest, the necessary restraint, cautious use of sedatives and hypnotics, and careful feeding. [The reader is referred to Section II, Vol. II, for a complete discussion of the treatment of alcoholism.—EDITOR.]

Korsakow's psychosis requires no treatment except what is called for by the other conditions present in alcoholic neuritis.

Arsenical Poisoning.—In cases due to poisoning by arsenic the treatment as to cause depends essentially on preventing the further introduction of arsenic into the system. There may be considerable difficulty in ascertaining the source of the poison, as in the early cases of the great epidemic in England, where beer was made with glucose which had been made with sulphuric acid, containing arsenic as an accidental impurity. Gowers (12) notes the danger of a far smaller amount of arsenic in beer than can be taken with impunity in a bromid mixture prescribed for epilepsy, and suggests as an explanation that alcohol augments and bromid restrains metabolism in the nerves. It might also be said that the impure beer contained two causes of multiple neuritis. When the source of the arsenic is known it may require a change of occupation, as in the case of one of my patients, a metallurgist whose neuritic pains returned whenever he resumed his work after apparent recovery. The ores worked with contained arsenic, but as he had not known them to affect others it required not merely the chemical proof of arsenic in the urine, but also several returns of the pain to convince him that they were really the cause of his trouble.

Not a few cases of herpes zoster and some of multiple neuritis have been caused by the medicinal administration of arsenic. This has generally been in severe or obstinate cases of chorea in which long-continued administration or large doses have seemed necessary. In other cases it has been due to the patient ignorantly continuing the same prescription long after the physician, who has forgotten all about it, would have discontinued it. Every prescription for this or any other dangerous remedy should carry a written or printed prohibition to refill it after a certain date.

When the absorption of arsenic is stopped elimination usually goes on steadily without any special treatment. It may be hastened, however, by the administration of laxatives and iodid of potassium. As in mercurial poisoning the iodid should at first be given in very small doses in order to avoid putting too much of the metal in the circulation at once.

I have seen less than ten grains of iodid very greatly aggravate the pain of arsenical neuritis, which subsided when the patient, on his own account, discontinued the remedy, and promptly recurred when it was resumed. Only on dropping to two-grain doses, and gradually increasing, was it well borne.

Lead Poisoning.—In neuritis caused by lead the patient's occupation and habits usually leave no doubt as to the source of the poison. But in some cases, even when the line on the gums or the presence of lead in the urine makes the cause certain, the mode of its introduction is very hard to determine. In these cases drinking water, medicines, especially hair restorers and other cosmetics, and all the substances with which the patient is habitually occupied must be under suspicion until proved innocent or the source of the poison is definitely known. An infinitesimal amount regularly absorbed for a long time will eventually bring disaster. The old books tell of a seamstress who was poisoned by habitually biting thread in the glazing of which lead was used.

If the patient's occupation involves the use of lead, as in painting, the danger may be minimized by carefully cleansing the face, hands, and nails, especially before eating, avoiding work in which the air breathed may be contaminated, as in painting ceilings or working in the dust of lead ores, frequent bathing of the entire body, wearing none but clean clothing next the body at any time, changing clothing as soon as work is over for the day, and using the sulphate of sodium or magnesium as a laxative. But if the nerves are already involved at least an interruption of work will be necessary.

To favor elimination potassium iodid should be given, beginning with small doses and gradually increasing. As gout and nephritis are frequent complications of plumbism the action of the kidneys, particularly in the elimination of uric acid, should be favored in every way possible.

Diabetes.—The causal treatment of multiple neuritis due to diabetes includes all the dietetic measures that may be used to combat this disorder of metabolism, which is necessarily exceedingly grave when it causes neuritis. These measures are systematically considered in another part of this work.

Infections.—The treatment of various infections which may cause multiple neuritis is given in special articles. The occurrence of neuritis calls especially for elimination and support. Gowers strongly recommends tincture of iron in doses of 20 to 30 minims.

TREATMENT IRRESPECTIVE OF CAUSE

In discussing the treatment of multiple neuritis itself, irrespective of cause, it is convenient to follow Goldscheider and divide the disease into three stages: 1. The stage of advancing muscular paralysis. 2. The stage of arrest. 3. The stage of convalescence and regeneration.

Diet.—In all forms of multiple neuritis and in all stages the food should be as abundant and as rich in proteins and fats as the patient's powers of digestion will permit. It should be given in moderate quantity from four to six times daily rather than in a large amount three times a day. In the diphtheritic form special difficulties in feeding the patient are likely to be encountered because of the frequent paralysis of the muscles of deglutition. If only the palate is paralyzed so that the principal difficulty is regurgitation of food through the nose the patient may get along fairly well with semisolid foods, such as custards, puddings, eggs in various forms, cottage cheese and pap; these being easier to swallow than either solids or liquids. If the pharynx or epiglottis is paralyzed the taking of food of any kind becomes both difficult and dangerous. When enough can no longer be swallowed the nasal or esophageal tube must be used without delay, as a fatal weakness of the heart and respiratory muscles is likely to come on rapidly as soon as the amount of food is insufficient. On account of this danger the patient's emotional state is of the greatest importance and the physician must use his utmost skill and tact to conduct the feeding process so as to occasion the least possible distress or alarm. Rectal feeding of peptonized milk may be used to supplement that by the stomach, but is far inferior and can supply only a fraction of what is needed to make good the losses of the body.

Elimination.—If an abundance of food is to be taken in a disease caused by a poison free elimination is obviously necessary. Small doses of calomel with tonic and saline laxatives should be used to secure sufficient action of the bowels, but without such a degree of purgation as will weaken or interfere with rest. As a diuretic salicylate of sodium is particularly useful in all gouty or rheumatic cases. It should not be given in solution, as in this form it always becomes repulsive in a short time on account of its effect on the mouth and pharynx. If given in tablet form immediately before food it will almost always be borne perfectly well by the stomach. Plenty of water should be taken for its diuretic action.

Tonics.—Strychnia is especially indicated on account of its influence in increasing appetite and digestion, its tonic effect on the heart and respiration, and its general tendency to stimulate motor neurons. Its dose is gm. 0.002 (1-30 of a grain) three or four times daily. If the heart is weak and rapid digitalis should also be given, gm. 0.05 to 0.1 (¾ to 1½ grains) of the powdered leaves, or 8 to 15 minims of the tincture, three or four times daily. If respiration is threatened atropia hypodermically, gm. 0.0007 (1-100 of a grain), will tend to sustain it. It may be given every four to six hours.

Rest.—In the first stage rest is by far the most important desideratum. Every muscular contraction presses upon and irritates the nerves, thus hastening the destructive process as well as increasing the pain. Even in the lightest cases, where it is very difficult to induce the patient to submit to confinement, there is a great probability that efforts to keep up as long as possible will prolong the disease. In all but the lightest cases rest should be in bed, and as nearly absolute as possible. If the heart and respiratory muscles are seriously weakened the patient ought not even to sit up in bed. This is especially important in the diphtheritic cases, where the danger of sudden death from cardiac failure is considerable.

Warmth.—In the first stage thermal rest is as important as mechanical rest. Cold is depressing, uncomfortable, and dangerous to the tissues. A hot-water bag or poultice, which is hot enough to be barely endurable by healthy tissue, is too hot for an inflamed nerve or its distribution, and involves the risk of blistering or even of deep sloughing. Moderate warmth as nearly equable as possible is most soothing and most favorable to subsidence of the inflammation. Warmth with moisture may be obtained by wrapping the limbs in cotton and covering this with oiled silk, rubber tissue, or paraffin paper, the whole retained by a roller bandage just tight enough to stay in place. If the natural warmth of the limbs thus bandaged is not sufficient external heat may be added cautiously by means of a hot-water bag or any convenient heated object, provided this is carefully insulated from the skin and is not hot enough to burn if the coverings should accidentally be displaced. Later, as the more acute symptoms are subsiding, dry warmth is preferable and may be secured by omitting any waterproof material from the bandages and changing the cotton as often as it is moistened by perspiration.

Posture.—In the most painful stage the position of greatest ease is naturally chosen: thighs slightly flexed and rotated outward, knees slightly flexed, feet at right angles to the legs, arms slightly abducted, elbows slightly flexed, arms between pronation and supination or slightly pronated, wrists between flexion and extension or slightly extended, thumb and fingers semiflexed. This posture is to be maintained without active effort by means of skillfully adjusted pillows of varying size. From the beginning the tendency to foot-drop and wrist-drop and to these deformities becoming fixed must be borne in mind. The feet may be supported in proper position by a large sand bag, nine inches in diameter, placed transversely, or by a board fastened across the bed. The slight pressure needed to support the feet should come upon the ends of the metatarsal bones. The feet must be protected from the pressure of the bedclothes either by the arrangement of pillows or by a specially devised wire frame. At first the sand bag or board will support the feet more comfortably than any dressing, but later it may be more convenient to use the apparatus devised by Gowers. As pain subsides the semiflexed position of the hips and knees should be changed to that of full extension, otherwise the resumption of walking will be much delayed by contraction of the flexor muscles. This change in posture causes a little distress at first, but with patient persistence can be managed without much discomfort. To prevent fixation of any of the joints passive motion should be begun as soon as it can be carried out without causing severe pain at the time, or more than slight pain persisting for a short time after the manipulation. The feet are to be dorsally flexed, the wrists and fingers extended, and so on, but all the motions must be carried out slowly and with extreme gentleness. If one manipulation causes a definite increase of pain, or makes the patient dread the next, it has been overdone.

Pain and Insomnia.—The measures already described tend strongly to relieve spontaneous pain, but in most cases enough will remain to require additional relief by means of analgesics or narcotics, as described in the treatment of neuritis in general. These should be used sparingly, but the relief should be adequate, especially at night. When sleep is prevented by pain opium or one of its derivatives is the best hypnotic.

When there is wakefulness, independent of pain, chloral, on the whole, seems to me to be the best hypnotic, and it may well be reinforced by a moderate dose of bromid. It will generally be best to make a skillful combination of drugs to meet the special indications.

In the early stage of acute pain and advancing paralysis massage and electricity ought to be omitted entirely. Bathing should be limited to the tepid sponging necessary for cleanliness.

Massage.—After the advance of the disease is arrested and pain has subsided massage should be begun. At first it should be merely a gentle superficial upward stroking of the limbs, but as tolerance is ascertained the rubbing may reach the deeper tissues so as to favor the flow of lymph and venous blood toward the trunk. The passive motions already begun can now be combined with massage made more vigorous. If any muscles, especially the calf muscles, show a tendency to shorten they should be stroked to favor their relaxation while being stretched by the appropriate passive motion.

Electricity.—Galvanic electricity may be used with advantage during the second stage. It is essential that each group of muscles as it is treated should be relaxed by posture and thus be free to contract. The pole on the muscles should be the one which causes the greater contraction, either negative or positive, the current should be slowly made and broken and strong enough to cause fairly vigorous action. The static spark has been especially recommended, but the practical inconvenience attending its use is generally too great.

Convalescence.—In the third stage electricity, massage, and passive movements are to be kept up until returning motor power and active exercises render them unnecessary. In sitting foot drop must still be guarded against by seeing that the ball of the foot is supported while the heel is free to drop. If the calf muscles still tend to shorten they can be stretched by attempts to stand and walk. As soon as the patient is able to walk at all they generally yield and improvement goes on rapidly. Warm baths favor relaxation. In only a few cases will section of tendons be necessary. The open air, tonics, food, recreation, and remedies to favor digestion and elimination will naturally be suggested. Finally, as in other cases of long-continued illness, convalescence can often be hastened and made more complete by change to some agreeable place in a climate which favors outdoor rest and recreation.

In the alcoholic cases the period of ultimate recovery should be utilized by the physician in explaining and urging upon the patient the necessity of total abstinence for the rest of his life. He must learn how, as a matter of habit, to ignore the existence of alcohol altogether, avoiding even the resolutions and protestations that he will not drink, because they awaken the dormant appetite.

LANDRY'S PARALYSIS

(*Acute Ascending Paralysis*)

This is a febrile disease, of acute onset, marked by flaccid paralysis beginning in the lower limbs and ascending, in the fully developed cases, through the trunk to the arms, then in the fatal cases to the diaphragm and the muscles of the tongue, pharynx, and larynx. Sensory loss is absent or limited to a slight dulling of sensibility below the knees. There is no severe pain and constitutional disturbance is comparatively slight. The muscles show no conspicuous atrophy or decided change in their electrical reactions. Control of the sphincters is retained. After death no gross lesion can be found in the cord or nerves.

It follows from this that the disease is different from myelitis, poliomyelitis, or multiple neuritis. It is a practical certainty that it is an infection whose toxin has a selective effect on the anterior horns or the motor roots, but the microorganism has not yet been identified.

In the present state of knowledge there is no efficient treatment, although we hope that bacteriology will indicate a remedy in the not distant future. In the meantime, on account of its tendency to sterilize the fluids of the body, it seems more rational to give hexamethylenamin than any other drug, gm. 0.5 (7½ grains) four times daily. Strychnia is also rationally indicated, gm. 0.002 (1-30 grain) four times daily. Strümpell (43) recommends aspirin or sodium salicylate, at the beginning in large doses. He also recommends mercurial inunctions used cautiously. Ergotin is well spoken of by Oppenheim (30), but regarded as useless by Buzzard. Counterirritation, by blisters or even the Paquelin cautery, has been recommended, but I would omit it. From the beginning the most perfect rest and comfort should be secured. The bowels should be opened and the catheter used if necessary.

When death comes it is through respiratory paralysis. Therefore, care should be taken to prevent or cure even a slight bronchitis. When respiration is embarrassed atropin should be added to the strychnin both to stimulate the respiratory center and to check bronchial secretion, gm. 0.0003 to 0.0006 (1-200 to 1-100 grain) four times daily. Toward the end life may be prolonged by artificial respiration and oxygen. When the danger to life is passed there is but little need of treatment but galvanic electricity, passive movements, and massage may be indicated to hasten convalescence.

POLYMYOSITIS

Excepting the familiar muscular rheumatism and trichiniasis, primary diseases of the muscles are rare and comparatively little known. Of recent years, however, cases representing a group of diseases have been collected and described under the general name polymyositis, the essential lesion being inflammation of a number of muscles. For a general account the reader is referred to Senator (40), Strümpell (43), Oppenheim (30), and especially Steiner (42). In all of these treatment will be found the least satisfactory phase of the subject.

The purulent form of polymyositis is a multiple acute inflammation of the muscles, beginning like an acute infectious disease and ending in the formation of abscesses in the muscles. The staphylococcus pyogenes aureus has been found far more frequently than any other germ, sometimes in a pure culture. The nonsuppurative forms include polymyositis hæmorrhagica, in which there are hemorrhages into the inflamed muscles; dermatomyositis, in which both the muscles and the skin and subcutaneous tissue covering them are inflamed; and neuromyositis, in which some of the nerve trunks are involved.

The treatment of all of these conditions is substantially the same as that of multiple neuritis, the modifications depending on obvious indications. In the first stage rest as complete as possible and elimination through laxatives, diuretics, and diaphoretics are the important objects. Pain should be combated, first with salicylates, as in acute rheumatism. Aspirin is the favorite salicylate now on account of its diaphoretic property. Other pain-relieving drugs are to be used on the same principles as in neuritis. When the fever is high cool sponging should be employed, later warmth will be better. In the purulent form pus should be evacuated as soon as detected. Whether a vaccine can be employed to advantage in these cases must be determined by the degree of absorption that is already going on, and the probable power of the patient to respond to an additional demand on his immunizing forces. The indications as to posture, passive motions, massage, electricity, and care during convalescence are precisely the same as have been considered in detail under multiple neuritis.

[I have seen one case of dermatomyositis which recovered after most liberal inunctions of Credé's ointment.—EDITOR.]

OCCUPATION PARESES

The cases whose treatment is here discussed do not include all of the occupation neuroses, which are defined by Oppenheim as "disturbances of innervation which occur only in certain complicated movements acquired by practice, the muscles responding to the will in every other action." The disturbance of innervation may be manifest as spasm, pain, tremor, or paresis of the overworked muscles. Only the pareses are here considered, and to them the latter part of the definition does not apply, for the paretic muscles do not respond to the will in every other action. It is indeed a question whether this part of the definition applies to any case of occupation neurosis, but it certainly does not apply to true paretic cases (Poore, 35; Paul, 32).

The essential cause of an occupation paresis is long-continued overwork of certain muscles, usually of the hand. The muscles that most frequently suffer are those kept in continuous tonic contraction in order to grasp an implement or maintain a posture. Thus in writer's palsy it is the abductor indicis and opponens pollicis which maintain a continuous opposing pressure on the pen, that are most often affected; in dancers it is the calf muscles, which maintain the tiptoe posture; in piano players the extensors of the wrist, which must continuously maintain the hand in proper position in opposition to the tendency of the active flexors of the fingers to flex the wrist.

Less frequently the paresis is in a muscle which contracts and relaxes rhythmically an enormous number of times, as in the weaver mentioned by Oppenheim (30), who "showed symptoms of paralysis of the right triceps due to the fact that he had to extend the forearm about 20,000 times a day." In the same way working a pedal, as in professional bicycle riding, may cause paresis of the calf muscles.

Combined with overwork of the muscle there is often an injurious degree of pressure exerted upon it or its nerve, for example, by the tool used pressing on the palm of the hand, or by the ulnar side of the hand or the elbow habitually resting on the table. In such occupations as beet growing and potato digging, which require the long maintenance of a stooping or kneeling posture, not only are the muscles overworked, but nerves are both pressed upon and stretched. If "crick in the back" is added to peripheral disability the sacroiliac joint is probably strained, and the sciatic nerve irritated within the pelvis. (See Sacroiliac Sciatica.)

In addition to the overwork and local pressure or tension a systemic predisposition is often found, such as alcoholism, gout, rheumatism, lead poisoning, semi-starvation, anemia, deficient assimilation, or a neurotic constitution. Weakness of synergic muscles, as from a former poliomyelitis, naturally predisposes to an occupation paresis.

Although the anatomical changes in occupation pareses have not yet been ascertained and chronic fatigue has been regarded as an explanation the clinical features make it highly probable that there is organic disease in all of them. That many are special forms of neuritis is indicated by the accompanying pain, tenderness, possibly slight sensory loss, diminution of faradic irritability, and sometimes distinct muscular atrophy. The morbid change may be in the fibers of a nerve trunk, but are generally to be looked for in the finest terminal fibers or the motor end plates. Oppenheim thinks there is also a myositic form. In the pareses other movements than those peculiar to the occupation which require prolonged or often repeated action of the affected muscles are also impaired.

The statement of the causes and probable nature gives the indications for treatment, which are:

1. To rest the affected muscles and their nerves.
2. To free them from pressure and tension.
3. To improve the general health and treat any constitutional defect or toxic state.

4. To hasten the restoration of the paretic muscles and their nerves.

Rest.—Overwork being the essential cause, rest of the affected muscles is the essential remedy. I have known typewriters and musicians, on the first appearance of the defect, to try to make up for it by practicing with extraordinary zeal, thus greatly accelerating the pathological change. As a rule the demand for rest is absolute. In a few cases, not yet far advanced, in which unfavorable systemic influences can be removed, and important local factors can be eliminated by a change of posture or a better method of grasping an implement, the practical needs of the patient may warrant the continuance of a reduced amount of work. In most cases, however, the occupation must be given up until recovery is complete, and even then it is highly desirable to continue in some other means of livelihood. Other forms of activity requiring the coöperation of the affected muscles must also be given up or as far as possible restricted, but ordinary use of the limb, exercising other muscles and joints, is not objectionable. A patient with writer's paralysis, for example, ought not to take up sewing, knitting, or wood carving, because all of these require prolonged tonic contraction of the opponens pollicis and abductor indicis, as writing does, but he could use the typewriter or play the piano because in these activities other muscles are used.

In the early stage, and as long as there is any local pain or tenderness, electricity and massage are as strictly contraindicated as work.

Local Injury.—Giving up the harmful occupation will generally remove any aggravating pressure or tension, but a careful examination of the muscles, tendons, joints, and nerves should be made with a view to discovering and removing any inflammation, tumor, or other local disease. Round shoulder may be an important factor (see Brachial Neuritis). If the defect is in the sciatic distribution the sacroiliac joint should be examined and if found relaxed an appropriate belt or other support should be used.

Systemic Causes.—Just as in cases of neuritis or neuralgia every effort should be made to improve the general health. It is especially important that food should be sufficient in quantity and quality. Strychnia is of great value as a tonic for the nervous and digestive systems. Iron, and perhaps moderate doses of arsenic, are of course indicated in anemia. The endogenous wastes must be eliminated and the bowel and kidneys must be kept sufficiently active. In gouty or rheumatic conditions sodium salicylate or one of the numerous other salicylates should be administered. Of the exogenous poisonings, alcoholism is the one most frequently found. The neurasthenic state may aggravate the defect and require careful treatment.

Massage and Electricity.—After all acute symptoms have subsided massage may be begun. It must be very gentle at all times, for the nerve endings and muscle fibers are very delicate structures whose normal vulnerability has been greatly increased by the disease. Very gentle rubbing, merely sufficient to facilitate the return of venous blood and lymph from the muscle, is all that should be advised or allowed.

Galvanic electricity should be used, as in neuritis, to improve the nutrition of the muscles. The negative electrode is applied to the muscle, the positive elsewhere on the limb, or in any convenient area, and the current slowly interrupted with just enough strength to cause moderate contraction on closure. The posture must be such as to relax the muscle being treated. The faradic current should not be used.

REFERENCES

Since Chapters II, III, and IV are so closely related, the references for all three chapters have been combined in a single list at the end of Chapter IV, p. 360.

CHAPTER IV

THE NEURALGIAS

HOWELL T. PERSHING

NEURALGIA

The term neuralgia has been used so loosely to designate pains having a very different origin from those of true neuralgia, and hence requiring very different treatment, that it is necessary at the outset to understand what is meant by it.

Pain, although it may be in the course and distribution of nerves, if kept up by organic disease that can be recognized clinically, ought not to be called neuralgia. This excludes the pain caused by unhealed wounds, mechanical pressure, new growths, inflammation, or visible degeneration acting on the nerve in question. Hence the directions to be given for the treatment of neuralgia are not intended to apply to the pains of tabes, herpes zoster, or neuritis, still less to those caused by disease of bones or joints. But if pain of the paroxysmal neuralgic character persists after an original organic cause has been removed, or if an irritation acting only from a distance, reflexly, causes such a pain, we must for purposes of treatment regard it as neuralgia. On the other hand, not all of the functional nerve pains are to be called neuralgia. The symptomatic pain of certain cases of occupation neurosis, migraine, epilepsy, or hysteria may closely simulate that of neuralgia. To apply general conclusions derived from the apparently successful treatment of certain cases of these neuroses to the treatment of true neuralgia is sure to lead to disappointment, and yet this has often been done, and it accounts for the presence of many worthless drugs on the list of remedies for neuralgia. Conversely to treat these neuroses in the way that would be appropriate were the case one of true neuralgia is a gross error which, nevertheless, has often been committed.

Neuralgia is here understood to mean pain felt definitely in the course or distribution of certain nerves, occurring in paroxysms, with intermissions or at least marked remissions, and not directly due to recognizable organic disease or to another neurosis such as migraine, epilepsy, hysteria, or occupation neurosis. It is not claimed that any definition will enable us always to discriminate between true neuralgia and the various organic and functional diseases that may resemble it, but the distinction must be kept in mind in discussing the value of different modes of treatment.

Neuralgia may affect any sensory nerve, so we have different neuralgias named according to the location of the pain. All of these are nearly alike in the broad features of etiology and treatment, and to avoid repetition the measures generally applicable will be discussed first, those specially applicable to certain localities coming afterward. Trigeminal neuralgia is the most typical form, and what is said of neuralgia in general applies especially to it.

TREATMENT AS TO CAUSE

Local Causes.—After a thorough anamnesis and general examination a careful and systematic search for local causes of irritation is to be made. Such a cause is more likely to be found in the distribution of the branch most affected, but it may be in that of another branch, or possibly even in that of another nerve. In the eyes inflammation of any part should be looked for and the state of refraction and muscle balance ascertained. The condition of the nose and its adjacent sinuses and of the external and middle ear should be investigated. But it is defect of the teeth that is most likely to be in causal relation to the pain, and they should be examined carefully in systematic order. It will be necessary to get Roentgen ray plates if there is any question of faulty eruption. The physician must be sure that the dental examination is thorough. Patients have assured me that there could be nothing the matter with the teeth, as the mouth had just been thoroughly examined, and yet on sending them to a dentist of my own selection most important lesions have been found.

If a source of irritation is found it should be carefully considered and removed. In some cases, especially the more recent, less typical, and less severe ones, this alone may effect a cure, but in the older cases and those of typical *tic douloureux* not too much should be expected. On the whole it seems to me that in this disease the curative effect of removing peripheral irritations has been greatly exaggerated. A few striking but exceptional cases have been the basis of a generalization without taking into account a far greater number that would point to a different conclusion. Sound teeth should not be extracted no matter how definitely the pain is referred to them, nor how decided the patient may be in wishing to have them out. If a sound tooth is extracted the pain persists unchanged or increased, or after a brief inhibition returns, probably worse than ever, and may still be referred to the socket of the absent tooth years later. Similarly a certain degree of caution and skepticism should govern in treating ocular defects supposed to be the cause of trigeminal neuralgia.

By all means let the eye be put in the most perfect condition possible, with a reasonable amount of examination and treatment, but it is not wise to lay too much stress on slight variations of refraction or muscle balance in a neurotic patient, and as a consequence to keep making changes in the glasses at short intervals. Local irritations in the viscera of the chest, abdomen, or pelvis are very difficult to interpret as possible causes of neuralgia, but the examination should be sufficiently thorough to detect them if present, and as far as possible they should be corrected.

Systemic Causes.—These are generally more important than the local or reflex causes. Anything that debilitates or poisons the system may be the principal cause of the excessive irritability of the sensory center causing the pain. The outlook is the more hopeful the more definite and adequate such cause may be, provided it is amenable to treatment. Anstie's famous epigram, "The pain is the cry of the nerve for better blood," may be true in either of two distinct senses: the cry may be for richer blood or simply for blood unloaded of its impurities.

The various forms of anemia are to be treated with a generous diet, iron, arsenic, and other tonics and regulation of the bowels. Arsenic should be given in small doses except in the more severe forms of anemia, and preferably as sodium cacodylate. Gowers warns against a vegetable diet. He says: "A good supply of animal food is of great importance for all but gouty subjects. I have known severe neuralgia to occur first on the patient commencing a purely vegetable diet, to disappear when meat was taken, and recur with severity at each of four attempts to return to vegetarianism" (13). The diet of all debilitated patients should be rich, not alone in proteins, but also in fats, of which butter, cream, and cod-liver oil are the best. Small doses of phosphorus have been of distinct advantage in a small proportion of cases.

Arteriosclerosis and the defective nutrition due to advanced age strongly predispose to neuralgia. They are naturally most resistant to treatment, but potassium iodid and the vasodilators, sodium nitrite or glonoin, do some good.

In patients who are run down rest is most important. Generally moderate activity with adequate periods of rest is better than idleness, but unfortunately many patients cannot rest, although having abundant time for it. In these restless, irritable patients small doses of opium or svapnia, gm. 0.01 to 0.02 (1-6 to 1-3 of a grain), three to four times daily, added to the tonic given and combined with enough aloin, gm. 0.002 to 0.005 (1-30 to 1-12 of a grain), to overcome the constipating effect, will be of great advantage. In some cases the systematic rest cure as devised by S. Weir Mitchell should be carried out.

Recent Rheumatic Cases.—In the acute cases of recent origin, apparently caused by exposure to cold or an accumulation of wastes of the uric acid class, warmth, both local and general, is indicated, together with moderate purgation, diuresis, and diaphoresis. Sodium salicylate or its equivalent among the many salicylates available acts almost as a specific. Aspirin is especially valuable in acute cases because it promotes free sweating, but it is more irritating to the digestive organs than sodium salicylate or salophen. Its dose is gm. 0.3 to 1.0 (5 to 15 grains) three or four times daily. The old-fashioned pulvis ipecac. et opii, gm. 0.65 (grs. x), at bedtime, with a hot foot bath, is appropriate at the beginning of such a case.

Gout.—The gouty, rheumatic, or uric acid diathesis is a very frequent chronic predisposing cause of neuralgia, and it is reasonable to suppose that the same chemical substances which commonly irritate the joints cause neuralgia by a direct or indirect irritation of sensory neurons. All such patients should avoid alcohol and foods rich in nucleins, such as sweetbreads, liver, and kidneys. The liver and intestine must be kept active and a salicylate should be given up to the limit of easy tolerance. I have found the salicylate of sodium far more useful than any other drug. Aspirin is highly efficient. If, however, there is doubt as to the patient's tolerance salophen can be given in the same dose as sodium salicylate, from four to six times daily, irrespective of food, with assurance that it cannot disturb digestion.

Other Intoxications.—Alcoholism, diabetes, lead poisoning, or arthritis deformans may be the systemic cause of neuralgia. They are treated in special articles in other parts of this work.

Infections.—Influenza is the most important of the acute infectious causes. It should be treated, like the acute rheumatic form, with the most absolute rest possible and with the greatest care to prevent a relapse, which is easily caused by going out before recovery is complete. Of the chronic infections malaria is especially apt to affect the supraorbital branch so that, in some regions, "brow-ache" is an old synonym for malaria. It should, of course, be treated by the prevention of mosquito bites and such doses of quinin and arsenic as may be necessary. Syphilis, although it is more apt to cause neuritis than true neuralgia, is a not uncommon cause of intercostal or supraorbital pains that cannot at first be distinguished from true neuralgia, and urgently demand specific treatment. One must remember, however, that neuralgia may exist in a syphilitic patient without being kept up by syphilis, and so mercury, iodid, and salvarsan must not be given too freely in a vain attempt to prevent the attacks. Pains caused by the parasyphilitic diseases, tabes and paretic dementia, are in a different category from active syphilis, on the one hand, and true neuralgia, on the other. Iodid of potassium in moderate doses may be of considerable service in patients with neuralgia who are not syphilitic. Arsenic also may be useful in patients who are neither anemic nor malarial; it should be given as sodium cacodylate.

The emotional condition of the patient is important. Grief, anger, fear, and other depressing emotions may precipitate attacks, and, conversely, exhilarating and cheerful influences may relieve or prevent them. Prof. W. B. Carpenter has told how attacks of severe trigeminal neuralgia often occurred about the time of his lecture on physiology, making him feel that it would be impossible for him to deliver it. He generally did deliver it, although with very great effort at the beginning, and as he became more and more interested the pain commonly disappeared. At the end of the hour it sometimes returned, but often it did not.

Many neuralgic patients are plainly influenced by climate, season, and weather. They suffer especially when storms are approaching from the west and in cold damp weather with rapid changes of temperature. When one can choose, a dry, warm climate is to be preferred, but in any climate the neuralgic patient should have an apartment that can be properly warmed.

Relief of Pain

Electricity.—Our profession is not unanimous in regard to the value of electricity in neuralgia. My own success with it, except in cases that have yielded readily to other lines of treatment, has been rather insignificant. Nevertheless, so many of the best observers testify to its value that it cannot be ignored.

The galvanic current is to be chosen in preference to either faradic or static electricity. It has been shown that it lessens the irritability of sensory nerves, and experience proves that it is the most useful of the three. The anode, about 4 cm. or 1½ inches in diameter for the face, soft and well moistened with warm salt solution, is gently applied to the seat of pain, while the cathode, which may be of any convenient size, is applied to an indifferent place, as the back of the neck or the chest. After the electrodes are properly placed the connection is to be made and the resistance of the rheostat slowly and smoothly diminished so as to increase the current very gradually until the desired strength is reached; then it is allowed to flow steadily for a few minutes and gradually reduced to zero before removing the electrodes. At the first sitting a maximum current strength of one milliampère for each square inch of skin touched by the active electrode, or for each six square centimeters, will be sufficient. At later sittings the current density may be two or three times as great and the time of application may be increased. Where the painful area is large a correspondingly larger active electrode should be used and the current made stronger in proportion to its area. In general the weaker currents applied for a long time, ten to thirty minutes, succeed best. This treatment should not be undertaken without a suitable rheostat and milliammeter. Sudden variations or excessively strong currents may very greatly aggravate the pain, and even its careful and skillful employment occasionally does harm.

The more sensitive the patient is to peripheral excitation, as in talking, eating, or touching the face, the greater the need of caution.

The faradic current is generally not suitable for the treatment of neuralgia, but one thoroughly skilled and experienced may sometimes use it to advantage. With very rapid interruptions and the high voltage obtained by a very long secondary coil a fine wire brush may be applied to the skin of the sensitive areas as a counterirritant, or a weak current may be applied to the nerve through an electrode or, better, through the physician's hand. With the modern high-frequency current which is a development of the faradic coil I have had no experience. It lowers blood pressure, and therefore might be of special value in cases associated with arteriosclerosis. Static electricity may be used as a breeze, or sparks may be used as a counterirritant. Its incidental psychic effect may be valuable.

In the inveterate cases of tic douloureux, where many remedies have been tried without success, I do not think it worth while to try electricity. If there is a possibility that the pain is that preceding an eruption of herpes zoster electricity, together with all irritating applications, should be avoided; otherwise the eruption may be attributed to an error in treatment.

Drugs to Relieve Pain.—These are always necessary at some stage of the case, and must often be employed in advance of any effort to remove causes. Their justification is not merely the relief of present suffering, although this would generally be sufficient; when successful they at least tend to prevent future attacks and so contribute toward a possible cure.

One of the coal-tar analgesics should first be tried. Acetphenetidin (phenacetin) is perhaps the best of these, and may be given in a single dose of gm. 0.50 to 1.0 (7½ to 15 grains), maximum in one day gm. 2.0 (30 grains). The larger doses are often necessary, and Byrom Bramwell has given as much as gm. 2.0 (30 grains) at once. Such doses are not devoid of danger unless the smaller ones have first been tried and their effect upon the heart and the condition of the blood carefully noted, with due regard to the age and general condition of the patient. Nevertheless this is a disease in which well-considered and carefully guarded risks are often justifiable, and remedies of this class carefully used are not so dangerous as many others, for example, aconitin or the larger doses of morphia. The maximum dose must not be repeated within at least twelve hours. The danger of failure of circulation is minimized by rest in bed and the administration of strychnia or quinin.

Instead of acetphenetidin antipyrin may be given in twice its dose, gm. 1.0 to 2.0 (15 to 30 grains), maximum in one day, gm. 4.0 (60 grains), subject to the same precautions, or acetanilid, gm. 0.3 to 1.0 (5 to 15 grains), maximum in one day, gm. 1.5 (23 grains). Salipyrin and pyramidon have a similar action and may be used instead. The dose of salipyrin is the same as that of antipyrin, of pyramidon the same as of acetanilid.

Butylchloral hydrate has been strongly recommended by Liebreich (23) as having a specific anesthetic effect on the trigeminus in doses too small to affect the heart or respiration or even to cause sleep. Others have controverted his statement, but Oppenheim has sometimes found it sufficient in small doses. It may be given as follows:

℞

Butylchlorali, ...	5.0	ʒi, m. xv.
Glycerinæ, ...	15.0	ʒiv
Alcoholis, ...	15.0	ʒiv
Aquæ, q. s. ad., ...	60.0	℥ii

M. S.—ʒi in water every ten minutes until relieved or six doses are taken.

Aconitin, the crystallized alkaloid of aconite, has been successfully used by some physicians. It is given in doses of gm. 0.0001 (1-640 of a grain) at first every half hour until four doses are taken. If this is not sufficient it may be cautiously increased each day until the maximum daily quantity of gm. 0.002 (1-32 of a grain) has been reached. The patient should rest in bed and the effect on the circulation should be most carefully observed. There is no doubt that given in this way aconitin may, for a time at least, control even severe tic douloureux, but it is one of the most deadly poisons known, and these doses, even though cautiously administered, are somewhat dangerous to life. I have never had the courage to give it a thorough trial and cannot recommend it.

If some of the foregoing remedies are not sufficient an opiate should be added to the coal-tar analgesic. The safest, although the least efficient, is codein. It very slightly increases the danger, but will often do good service in doses of gm. 0.03 to 0.13 (½ to 2 grains). If this is not sufficient the extract of opium, gm. 0.02 to 0.07 (1-3 to 1 grain), or morphia, gm. 0.01 to 0.03 (1-6 to ½ grain), should be substituted for the codein. If the pain defies these milder measures, rather than resort to the very large doses of opium, such as Trousseau recommended in the worst neuralgia, it will be better to give morphia hypodermically until a more radical treatment is carried out. The pain antagonizes the effect of morphia so that increasing and ultimately very large doses may be necessary, but the initial hypodermic dose must be small, gm. 0.006 to 0.01 (1-10 to 1-6 of a grain). The patient must, of course, not have the syringe in his own hands, and if he happens to be a physician he should be required to give his own hypodermic case into the care of some one else, no matter how inconvenient it may be. When morphia is required daily it is time to decide upon more radical treatment.

Aside from the drugs which fulfil a distinct causal indication, and those already mentioned, very many others have been recommended as valuable in the treatment of neuralgia. Bernhardt remarks that a writer loses courage as soon as he attempts to make even a brief mention of them. Vanlair gave over 150 in the first edition of his book, in 1866, and the number has been greatly increased since then. The best that can be said of most of them is that in a very small proportion of cases they have seemed to be helpful. Many, no doubt, owe their recommendation to having been used in cases of hysteria simulating neuralgia, or to having been administered in true neuralgia when a remission was about to occur from other causes.

Unhappily there are many cases of neuralgia in which the pain has returned again and again in spite of all medical treatment. This has naturally led to surgical operations, especially on the trigeminus, as its single motor branch is so far separated from the three sensory branches that the sensory functions can be destroyed without serious motor loss.

CUTTING OPERATIONS ON THE TRIGEMINUS

Simple stretching or cutting of the nerve trunks has generally stopped the attacks, but in most cases for only a few months, sometimes for a year or two. Until a few years ago the best operation was the avulsion method of Thiersch. In it the operator cuts the first or second branch at its exit from the supra- or infraorbital canal and, in order to prolong the period of relief, tears out as much as possible of each of the ends by catching it with forceps and winding the nerve trunk about them, thus removing a large part of the trunk and its branches and delaying the period of regeneration. The third branch has been dealt with in the same way at the mental foramen, or more effectually by making a small trephine opening in the ramus of the jaw, so as to expose the nerve in the inferior dental canal near its entrance. On account of the tendency of the pain to shift from the branch operated on to the next previously sound one it was often deemed best to operate on two or all three of the branches at once, even when only one seemed to be affected. Then, in order effectually to prevent regeneration, the operation was supplemented by plugging the foramen or canal with silver or another metal. Even after this apparently thoroughgoing operation, done by the most experienced operators, permanent relief was seldom secured, and when the attacks returned the only prospect of cure was in the final operation for the removal of the Gasserian ganglion.

The avulsion operation of Thiersch is described because of the place it has until recently held as the next resort after the failure of drugs and electricity, but it ought, I think, to be entirely abandoned. Alcohol injections will naturally be tried first, because they are easier for the physician and less formidable to the patient, can reach the second and third branches central to their first subdivision, give a longer period of relief, can easily be repeated, and do not compromise the success of any other operation that may ultimately be necessary. The avulsion operation, on the other hand, even when most thoroughly done and the canals plugged, gives a shorter respite, which becomes still shorter with repetition, cannot easily be repeated and, by leaving scar tissue about the eye, may imperil the result of the Gasserian operation that may afterwards be necessary. (Cushing, 8.) If the injections have been thoroughly tried and have failed it is safe to say that avulsion will not succeed.

Injections for Trigeminal Neuralgia

It is not an exaggeration to say that the treatment and prognosis of severe trigeminal neuralgia have already been revolutionized by the improved and simplified methods of injecting alcohol into the nerves. The evolution of the method has been gradual, yet rapid, and constitutes one of the most gratifying advances in treatment.

Almost from the introduction of cocain as a local anesthetic Gowers has insisted on the great relief to be obtained in neuritis and neuralgia from its hypodermic use. He has employed it in the distribution of the nerve and especially in the most painful areas as close to the nerve trunk as possible. Although the relief was only temporary and the injection had generally to be repeated once or twice daily he believed it helped toward a permanent cure. Others no doubt would have used cocain more freely in this way were it not for the danger of the cocain habit.

In the effort to get more lasting relief a number of more or less destructive substances were tried, including chloroform, ether, carbolic acid, and osmic acid. All of these had some success, and the injection of a one to two per cent. solution of osmic acid, in the hands of Bennett (1) in England and Murphy (24) in this country, was a distinct improvement on the operations of section and avulsion which it replaced. Relief was generally prompt and lasted for months or even years. Its disadvantages were that it was still necessary to do a formal operation to expose the nerves and that the osmic acid caused necrosis of any tissues it touched, even bone, thus favoring subsequent infection and suppuration.

In 1903 Schlösser, of Munich (37), described a method of injecting the branches of the trigeminus with alcohol at their exit from the cranium. He had tried injections at the supraorbital, infraorbital, and mental foramina and, thinking he would be more successful if the nerve trunks could be reached central to their important branches, he devised a method of injecting the third branch at the foramen ovale, the second at the foramen rotundum, and the lacrimal and frontal branches of the first as they enter the orbit at the sphenoidal fissure. He used a specially constructed bent needle for each branch and introduced it through the mouth. Schlösser's method was practiced in France by Ostwalt (27, 28, 29), and has been followed by Kiliani (20) and Hauck (17) in the United States. The difficult technique of Schlösser was simplified and made much easier by Lévy and Baudouin (22), and their modification has been used with success by Harris (15) in England and was introduced in this country by Patrick (31), who was followed by Hecht (18) and Bodine and Keller (4). The method of Lévy and Baudouin has now been tested by many operators in all parts of the world, and there can be no doubt of its great value.

Solution: The following formula will serve for most cases:

℞

Cocainæ hydrochl., ...	0.2	gr. iii
Alcoholis (95 per cent.), ...	17.0	ʒiv, m. xv.
Aquæ, q. s. ad., ...	20.0	ʒv

If absolute alcohol is used only 16 c. c. or ʒiv should be taken. For very obstinate cases Lévy and Baudouin add 20 per cent. of chloroform. This solution, if injected into a nerve, attacks the axis cylinders chemically and causes complete degeneration of all axis cylinders and medullary sheaths peripheral to the injection, leaving only the neurilemma. It thus accomplishes just what section would do in preventing impressions from the periphery from acting on the irritable nerve centers. Some influence is also exerted centralward upon the ganglion cells, for in experimental injections into the nerves of animals these cells show chromatolysis, but whether this has anything to do with the relief of pain is uncertain. If the injection is not into the nerve, but near it, the same effect may be produced, but with less certainty. The alcohol is not destructive to other tissues, and experiments on rabbits and dogs show that even if large doses of absolute alcohol are injected into the veins nothing worse than drunkenness will result. The solution is strongly antiseptic and, while this should not cause any laxity as to asepsis, it affords a very comfortable additional security against infection. It is not necessary to expose the nerve, and the necessary punctures leave no scars. A general anesthetic is not necessary, as the burning pain caused by the alcohol is not hard to bear in comparison with a paroxysm of neuralgia, and lasts only for a few moments, after which there is complete local analgesia. The patient remaining conscious and reporting his sensations, the operator knows when the nerve is struck. It may require several trials, however, before the nerve is thoroughly infiltrated and the full degree of analgesia attained. These attempts may be repeated at intervals of a day or two. It is essential that every superficial area whose irritation can excite a paroxysm of pain be rendered analgesic, whether by deep or by superficial injections. The relief of pain is usually immediate and, if the nerve is well injected back of the origin of any painful branch, the relief lasts from a few months to a few years, on an average longer than that obtained by section and fully as long as that obtained by the use of more destructive substances such as osmic or carbolic acid. When pain recurs, as it will in the majority of cases, it is as easy to make the injection again as it was at the first, there being no change in the relation of the parts and no scar tissue. The relief obtained from succeeding injections seems likely to be longer than that from the first. Kiliani says: "My general impression is that the recurrences become rarer, their intensity less, and I expect that a number of these cases which have recurrences at all will eventually remain free altogether. On the other hand, it is my belief that, in a very few cases, the recurrent attacks are fully as severe and less prone to yield to treatment." Sicard (41) finds that the first deep injections usually give relief for twelve to eighteen months, and that with subsequent injections the period of relief is longer, in some cases being two and a half to three years.

Injections at the Superficial Foramina

Supraorbital Branch.—The supraorbital notch may generally be felt at the junction of the inner third with the outer two-thirds of the upper margin of the orbit. After cleansing the skin with alcohol and ether and perhaps painting it with iodin, the finger is placed on the notch and the needle of an ordinary hypodermic syringe, containing a sterile one per cent. cocain solution or its equivalent, is inserted above the finger as accurately as possible into the notch and a few drops injected. Then the syringe with the alcohol solution is used in the same way and five to ten drops injected. If not sure of the notch one should probe with the needle just above the margin of the orbit in the endeavor to find a possible foramen. If still not sure the alcohol can be injected a little at a time at a series of closely adjacent points on the bony margin.

Infraorbital Foramen.—Draw a line from the supraorbital notch to the second bicuspid tooth in either jaw. Under this line, about 8 mm. or 5-16 of an inch below the lower margin of the orbit, lies the foramen. The corresponding point on the skin should be carefully marked. The margin of the orbit should be felt with the finger, not only to locate it, but to prevent the possibility of the needle entering the orbit. The needle must enter the infraorbital canal obliquely so the syringe rests on the nose and points upward and outward as well as backward, the needle entering the skin below and inside the point marked. Some patience may be necessary to find the foramen; no force is to be used and the needle must not be delicate enough to endanger its breaking. Only enough cocain is to be used to control the pain until the nerve is found and the patient complains of the peculiar referred pain in the nose, upper lip, and incisor teeth. Then a little more of the cocain solution is to be injected and the alcohol solution follows. If the needle is certainly in the infraorbital canal ten to fifteen drops of the alcohol is amply sufficient. If the foramen is not found at the first trial a larger quantity, 2 to 4 c. c. (30 to 60 minims), may be deposited at different points on the surface of the bone around the foramen. This injection is often sufficient even in a very severe neuralgia of the middle branch. In one of my cases where I wished to get the alcohol deep into the canal, so as to reach the branch supplying the socket of the upper canine tooth, which was the seat of greatest pain, the alcohol was felt trickling down the throat, showing that it had passed into the antrum. No harm was done and the complete relief obtained has now lasted about nine months. Kiliani, in making an injection into this canal, got an annoying oculomotor paralysis which lasted three weeks. He explains this on the assumption that his needle pierced the periosteum separating the infraorbital groove from the orbit, and that the alcohol acted on the ocular muscles and end fibers of the oculomotor nerve. Such accidents can be avoided by not inserting the needle more than 5 to 10

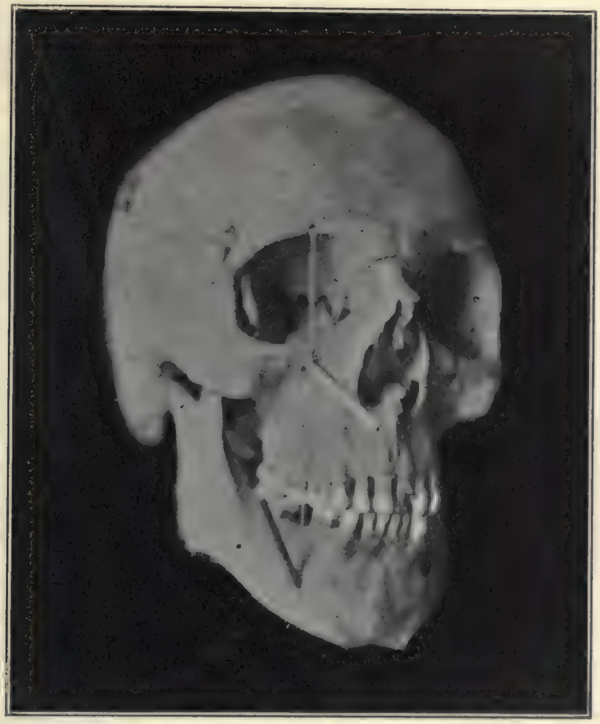

Fig. 1.—The string joining the supraorbital foramen and the mental foramen passes directly over the infraorbital foramen and opposite the second bicuspid tooth in each jaw. The direction in which the needle must point in order to enter the infraorbital or mental canal is clearly shown by the match projecting from each.

mm., 1-5 to 2-5 of an inch, into the canal and injecting the alcohol slowly, stopping when analgesia in the formerly painful area is complete, or if pain should be felt in the orbit.

The Mental Foramen.—This lies opposite the root of the second bicuspid tooth, or the space between the first and second bicuspids. In an adult whose teeth are retained it is halfway between the alveolar border and the base of the jaw, just below the cul de sac formed by the lower lip and the gum. In old people and those whose teeth have been extracted it is nearer the alveolar border and may even be directly on it. The needle should be entered from the outside, pointing obliquely downward, and a little inward and backward. The foramen is sometimes difficult to find.

Palatine Nerve.—If pain is referred to the roof of the mouth, and the middle branch is not injected in the sphenomaxillary fossa, it may be necessary to inject the palatine nerve where it issues from the posterior palatine canal. This is easily done by following the directions of Lévy and Baudouin (22): "The head resting on the occiput, the patient opens the mouth as wide as possible. A half centimeter (1-5 of an inch) inside the inner border of the alveolar arch, opposite the space between the first and second upper molar teeth, the needle is inserted. It (or the syringe) will rest on the lower lip. The region being cocainized, prudent search is made for the orifice of the posterior palatine canal. It is quite large and one will not be long in penetrating it." The point of the needle should pass obliquely upward and backward so as to reach the hard palate close to the alveolar border opposite the roots of the last molar tooth.

One or more of the injections so far described will often be quite sufficient to give complete relief even in severe and previously intractable cases, provided the hypersensitive areas, from which the paroxysms are excited, are rendered analgesic. They give all the relief that was formerly gained by the most thorough Thiersch or osmic acid operations and for fully as long a time, with the slightest possible inconvenience. They alone would constitute a very great improvement on the best treatment of ten years ago. Nevertheless, they are not sufficient for all cases, mainly because each division of the fifth nerve has important branches, supplying the eye, nose, teeth, cheek, or tongue, whose fibers, leaving the main trunk in a deep situation, escape the action of superficial injections. If their distribution is only moderately painful they may quiet down after the superficial injections, but if, as sometimes happens, the greatest pain is in the cheek, tongue, or jaws, one or more deep injections will be necessary.

Injections at the Cranial Foramina

For the deep injections I have adopted the simplified technique of Lévy and Baudouin (22), as introduced in the United States and described by Patrick (31).

A special needle is used, 10 cm. long, 1.5 mm. thick, bluntly beveled at the point and furnished with a stylet which, when inserted, makes the point a rounded one. It is marked in centimeters from the point up to five, so that its exact depth is always known. A fitting which screws on a hypodermic syringe makes a smooth taper fit with the needle.

I have also used a more slender needle 6 cm. long, screwing directly onto a hypodermic syringe, after having rounded the beveled point with a file and marking the distances 4 cm. and 5 cm. from the point. This has the advantage of allowing one to insert it with the syringe attached, and thus to cocainize as the point is advanced.

Lévy and Baudouin now use a slender, strong needle of very small bore, without a stylet, screwed to the syringe, 4 cm. long for the third division and 5 cm. for the second. The essentials are that the needle be strong, blunt enough to push an artery aside rather than pierce it, and of such a length or so marked that the operator can tell exactly how far the point is from the surface.

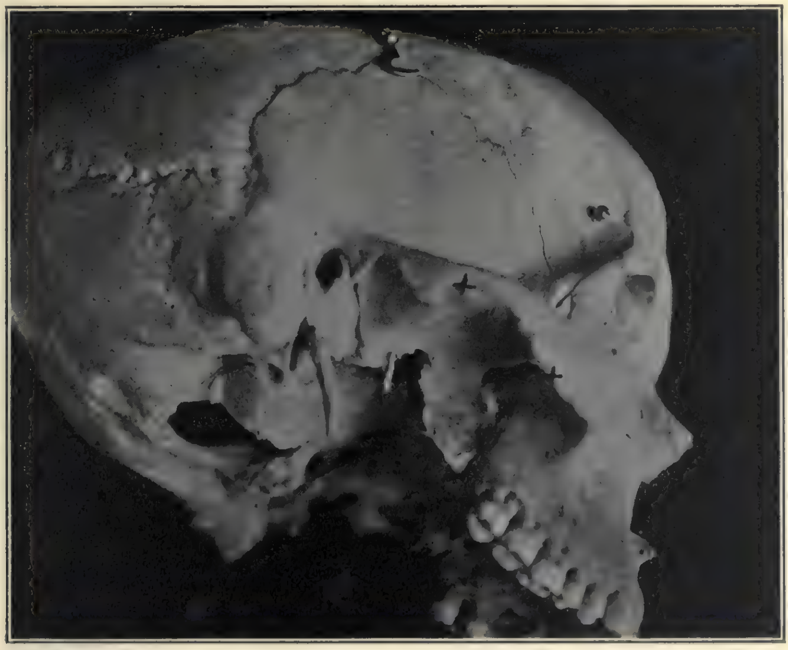

FIG. 2.—THE SKULL VIEWED OBLIQUELY FROM BELOW. The third branch of the trigeminus is represented by a black cord emerging from the foramen ovale just back of the external pterygoid plate. The middle meningeal artery is represented by a white cord in the foramen spinosum. The second branch of the trigeminus is represented by a black cord which can just be seen in the upper part of the sphenomaxillary fossa on its way from the foramen rotundum to the infraorbital groove. The points on the lower border of the zygoma for insertion of the needle are marked by crosses.

The Third Branch at the Foramen Ovale.—The needle is introduced just below the zygoma, 2.5 cm. (1 inch) in front of the descending root of the zygoma, which can be felt between the condyle of the jaw and the external auditory meatus. This point should be marked, cleansed with alcohol and ether, and painted with iodin. A little of a sterile one per cent. cocain solution should be injected into the skin and as deep as a hypodermic needle will reach. Then the skin is punctured with the point of a bistoury and the needle and stylet introduced, pointed nearly transversely, but also a little upward and backward, so that at a depth of 4 cm. the point of the needle will strike the base of the skull about 8 mm. back of a transverse line through the point of insertion. Considerable resistance is offered by the masseter and temporal fasciæ, which is to be overcome by moderate continued pressure and a boring motion. If the direction has been right, when a depth of 4 cm. is reached the point of the needle will lie in the foramen ovale and the patient will experience a paroxysm of characteristic pain in the lower jaw, lip, and tongue. The stylet should be withdrawn and the operator should wait a few moments to be sure that there is no bleeding through the needle.

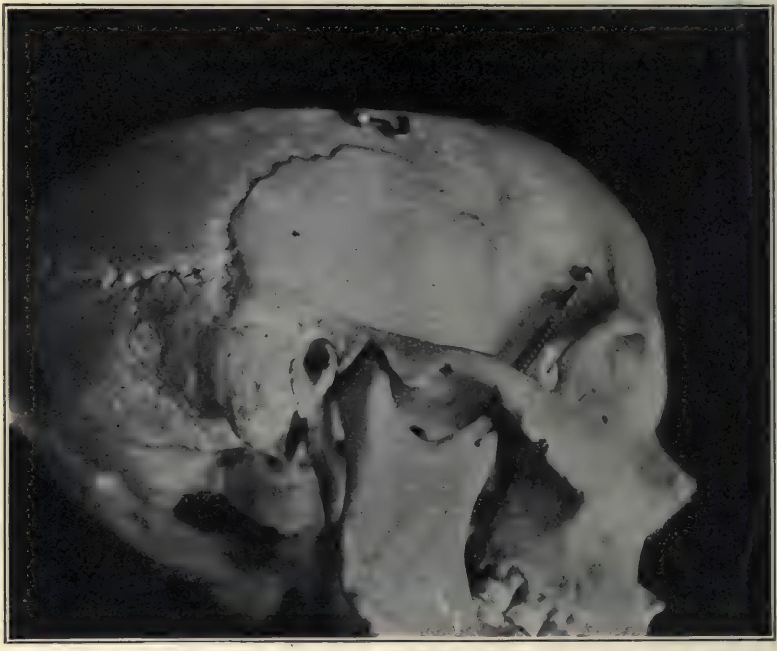

FIG. 3.—SAME AS FIG. 2, EXCEPT THAT THE LOWER JAW IS IN PLACE. The foramen ovale is just seen through the sigmoid notch. The black cord representing the second branch of the trigeminus is seen in front of the coronoid process. The black marks on the zygoma indicate the points for the insertion of the needle. In this skull there is plenty of room to reach the second branch but in some the coronoid process requires the point of insertion to be further downward and forward.

If a few drops of the cocain solution are now injected the pain promptly ceases, after which 2 c. c. of the alcohol solution is to be slowly injected, the needle allowed to remain in place a minute or two and then slowly withdrawn. Any oozing of blood is checked by pressure and the puncture sealed with collodion. If the alcohol is injected at once there is a burning sensation at the point of the needle and a sudden intense pain is felt along the nerve, both of which cease in a few seconds and are replaced by a numb sensation in the distribution of the nerve and some soreness at the seat of injection. The neuralgic pain is gone and the lower lip, cheek, gums, or tongue can be irritated without causing pain.

Instead of trusting one's knowledge of anatomy and sense of distance and direction to point the needle directly toward the foramen ovale, one can go more surely, according to the following instruction from Lévy and Baudouin (22):

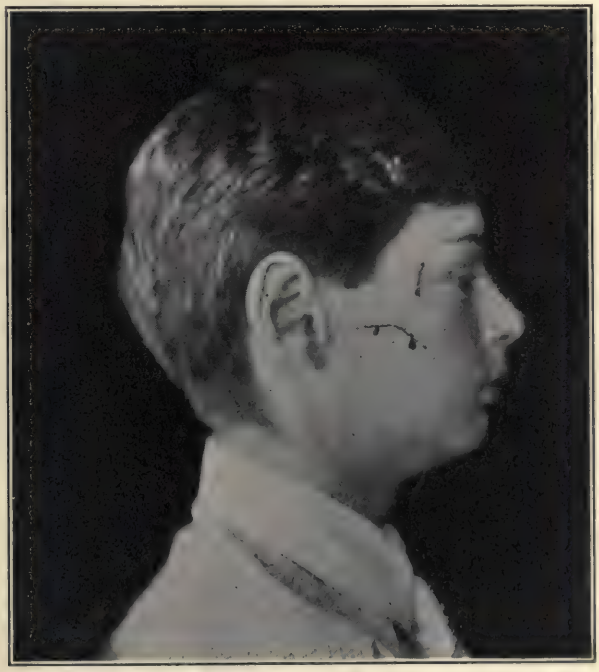

FIG. 4.—POINTS OF INSERTION FOR DEEP INJECTION OF THE SECOND AND THIRD BRANCHES OF THE TRIGEMINUS. The straight line back of the eye marks the posterior border of the frontal process of the malar bone and points to the place on the lower border of the zygoma where the needle should enter to reach the second branch. The point of insertion for the third branch is 2.5 cm. (1 inch) in front of the descending root of the zygoma or of the anterior bony wall of the external auditory meatus.

"The needle is inserted at the point indicated, but pointed slightly forward and upward. In this way it is always arrested at about 3.5 cm. by the bony surface which forms the cranial origin of the external pterygoid plate. It is necessary to go further back, but this cannot be done without withdrawing the needle some millimeters, in order to free it from the fibers of the external pterygoid muscle. The point is then slightly inclined backward and again pushed inward. If the same bony wall is again encountered, the maneuver must be repeated. But soon the needle is felt to clear the posterior border of the external pterygoid plate and sink deeper. It is now in the right place, either in line with the foramen ovale or immediately in front of it. This technique requires a strong resistant needle."

This method has the advantage of surely keeping away from the middle meningeal artery which lies behind and external to the foramen ovale, and also of minimizing the chance of paralyzing the motor root which passes through the posterior part of the foramen. It has the disadvantage of some additional pain unless the local anesthetic is injected as the needle advances.

It is important that the needle should not penetrate too far, not more than 4 cm. in an average adult skull or 4.5 cm. or 3.5 cm. in a very large or very small skull, respectively. Otherwise the Eustachian tube might be wounded or the pharynx punctured. In order to be sure of the depth to which the needle should penetrate in any skull, the following method devised by Offerhaus (26) may well be employed: The foramen ovale is in the same sagittal plane as the outer surface of the last upper molar tooth, at its neck, or the corresponding outer surface of the alveolar border of the upper jaw. Therefore, if one measures the transverse distance between the outer surfaces of the last molar teeth or of the extremities of the alveolar arch and divides by two, he will have the distance of the foramen ovale from the median plane. Suppose the distance measures 6.5 cm., then the foramen ovale is 3.25 cm. from the median plane. Now measure with calipers the distance from the point below the zygoma where the needle is inserted to the corresponding point on the opposite side. Suppose this is 14 cm., then the point of insertion is 7 cm. from the median plane, and the foramen ovale is 7 minus 3.25, or 3.75 cm. inside the point of insertion. As the needle is not quite perpendicular to a sagittal plane, about 2 mm. may be added, making 3.95 cm. the distance the needle should penetrate.

The dangers attending this operation are few and slight, and yet they must be borne in mind. The instruments, hands of the operator, solutions, and point of insertion of the needle must, of course, be carefully sterilized. A hematoma might possibly occur in a patient whose arteries and tissues are already in bad condition. Trophic disturbances, even gangrene, *might* occur, but I do not know that they ever have occurred from injection of this branch. A certain amount of ecchymosis is not uncommon, but it does no harm, and in due time disappears. Paralysis of the motor root of the trigeminus would cause deviation of the jaw to the sound side and confine mastication to that side. The muscles would doubtless recover in from a few weeks to six months or more. As a matter of fact, accidents of any kind have been exceedingly few.

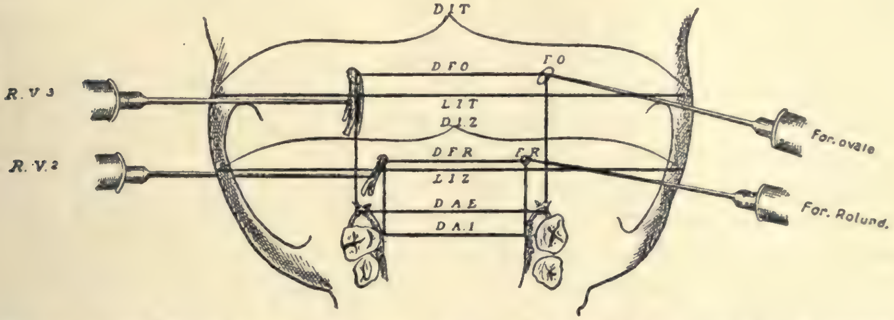

FIG. 5.—THIRD BRANCH OF THE TRIGEMINAL NERVE. D.F.O. = D.A.E. Needle to be inserted to a depth of ½(D.I.T.—D.A.E.) D.I.T. = Distantia intertubercularis. L.I.T. = Linea intertubercularis. D.F.O. = Distantia foram. oval. D.A.E. = Distantia interalveolaris externa.

SECOND BRANCH OF THE TRIGEMINAL NERVE. D.F.R. = D.A.I. Needle to be inserted to a depth of ½(D.I.Z.—D.A.I.) D.I.Z. = Distantia interzygomatica. L.I.Z. = Linea interzygomatica. D.F.R. = Distantia foram. rotund. D.A.I. = Distantia interalveolaris interna. (From Offerhaus, after *Jour. A. M. A.*)

The Second Branch at the Foramen Rotundum.—The line of the posterior edge of the frontal process of the malar bone, which can always be felt, is prolonged downward, and where it crosses the lower border of the zygoma the needle is to be inserted. In a normal skull this is just in front of the coronoid process of the mandible. The needle is to be inserted transversely, neither forward nor backward, but upward at such an angle that at a depth of 5 cm. its point will be on the same horizontal plane with the lower extremities of the nasal bones and about 3 mm. below the floor of the orbit. It passes through the pterygomaxillary fissure, between the maxilla and the external pterygoid plate, into the sphenomaxillary fossa, and if the inclination is correct it should at a depth of 5 cm. impinge on the superior maxillary branch, where it emerges from the foramen rotundum. Except for the difference in point of insertion, direction, and depth, the process is the same as already described for the third branch. When the nerve is reached characteristic pain is felt in the nose, upper lip, and upper teeth. If on withdrawing the stylet there is no bleeding, one c. c. of cocain solution should be injected, waiting to see if the eye is disturbed by it. If there is any diplopia or any other indication of disturbance within the orbit, the alcohol must not be injected at that point. If the orbit is not disturbed, from 1 to 2 c. c. (15 to 30 minims) of the alcohol solution is to be slowly injected. If this is successfully placed the distribution of the nerve will be numb and analgesic.

This injection is often more difficult to make than that into the third branch. The pterygomaxillary fissure may be very narrow and thus greatly restrict the range of movement within the sphenomaxillary fossa, even making it impossible to reach the foramen. The coronoid process of the mandible may come so far forward that the needle must be introduced further forward and lower, thus changing the direction of the needle and increasing the depth necessary.

Offerhaus (26) has devised a rule for finding the depth of the foramen rotundum like that for the foramen ovale. The foramen rotundum is in the same sagittal plane as the inner surface of the last upper molar tooth, at its neck, or the corresponding part of the alveolar border. If, therefore, the distance between the inner surfaces of the last upper molar teeth or the corresponding parts of the alveolar arch is measured, half of this distance is the distance of the foramen rotundum from the median plane. Suppose the distance between the teeth is 4 cm., then the foramen rotundum is 2 cm. from the median plane. Now measure with the calipers the distance from the point of insertion of the needle to the corresponding point on the opposite side. Suppose this is 13.5 cm., then the point of insertion is 6.75 cm. from the median plane and the foramen rotundum is 6.75—2 or 4.75 cm. from the plane of the foramen rotundum. If we allow an additional 2.5 millimeters on account of the obliquity of the needle, it should in this case penetrate to a depth of 5 cm. In the small skull of an undersized woman the depth should not be more than 4.5 cm.

There is more danger of accidents and complications in the deep injection of the middle branch than in that of the third. There is the same danger of hematoma if the vessels are diseased and trophic disturbances have been more serious. Schlösser (37) caused herpes gangrenosus in three of his cases. In one senile patient Patrick "caused a free deep hemorrhage," which was followed by necrosis of the hard and soft palate, with loss of a molar tooth. These lesions healed and the pain did not return.

The principal other danger is from the entrance of alcohol into the orbit, causing paralysis, especially of the sixth nerve. This is usually transient. Trophic disturbances in the eye, such as ulceration of the cornea, may also be caused. Aside from care in placing the needle, the best way to protect the contents of the orbit is, first to inject cocain, as already mentioned, and only when the eye is undisturbed to follow this up with alcohol. Considering the great number of deep injections done, accidents or complications of any kind have been very few, and mostly of a very slight or transient character. In the exceedingly small number of serious complications the patients would probably still choose the operation and all its results rather than to endure the torture of neuralgia.

Ophthalmic Branch

As this nerve divides while still within the cranium into its nasal, frontal, and lacrimal branches, the deep injection of its trunk is out of the question. The nasal branch cannot be reached without damage to the important motor nerves of the eye. Lévy and Baudouin (22), "in the rare cases in which injection at the supraorbital notch does not give good results," have injected the frontal and lacrimal branches by passing the needle along the external wall of the orbit, beginning at the junction of the external orbital process of the frontal bone with the malar. The depth is limited to 3 cm. and no important organ is touched. One naturally shrinks from invading the orbit with alcohol, and fortunately severe cases are far less frequent in this than in the other two branches. The supraorbital injection is so easily done and is generally so efficient, especially if pain in the middle and third branches is controlled, that, for the present at least, the orbit can be let alone.

Schlösser's Deep Injections

So far the Lévy and Baudouin method seems certain to give relief, for a time at least, provided the nerves are properly reached and infiltrated. In case the easier route is not practicable Schlösser's more difficult operation should be tried. It is possible, too, that thoroughly executed Lévy and Baudouin injections might fail and the original Schlösser method succeed, because in the latter the foramen ovale is approached from below, and the needle can be passed through the foramen into the Gasserian ganglion itself.

SURGICAL OPERATIONS

Although there is good reason to believe that many patients heretofore incurable by any means short of a Gasserian operation will at least be kept comfortable by the alcohol injections, it is not to be expected that all will be so fortunate. Kiliani (20) has been quoted as saying: "It is my belief that in a very few cases of treatment by injections the recurrent attacks are fully as severe and less prone to yield to treatment." We may hope that such cases will be only a very small proportion of the whole number, but, when the alcohol injections definitely fail to give relief, and the case is one of true neuralgia, not of hysteria on the one hand nor of organic cerebral disease on the other, a radical operation will be advisable. The pain impels patients to accept any risk of death or mutilation that affords a chance to escape from it, and surgeons are justified in performing the most desperate operations that promise relief, provided the safer measures have failed.

Ligature of the carotid artery promises too little and is too serious an operation to be recommended.

Jaboulay has excised part of the skull over the cortical sensory centers on the side opposite the pain in order to relieve them of pressure. Although these centers are undoubtedly the ultimate seat of the pain, there is nothing to show that it originates in them, while there is much evidence against such a supposition. This operation is a grasp at only a very remote chance of relieving pain. It might give temporary relief in the rare cases in which neuralgic pain is a symptom of tumor pressing upon the trigeminus.

No doubt new operative methods and combinations will be devised for the most obstinate cases. Lévy and Baudouin's method injects the second and third branches just outside the cranium, but leaves the Gasserian ganglion intact. Schlösser's original method of approaching the foramen ovale from the mouth, following the posterior edge of the external pterygoid plate, has the advantage that the needle can be passed through the foramen a measured distance into the ganglion, which can thus be partially destroyed. If, in addition to this, by using the old operation through the antrum, originally devised for the destruction of the spheno-palatine (Meckel's) ganglion, the needle were passed directly backward through the foramen rotundum for just the right distance, all of the ganglion except the ophthalmic portion could be destroyed by alcohol. This is all that was done by gasserectomy until recently, and the injection from both directions would be a far less formidable operation. Possibly the whole ganglion or its entire sensory root could be destroyed. How much danger of trophic disturbance in the ophthalmic distribution there would be could not be told in advance, but it seems probable that it would be no greater than in gasserectomy.

Intracranial Gasserian Operations.—These are the final resort. The first Gasserian operations consisted in cutting the second and third branches at their entrance into their foramina and tearing out the corresponding two-thirds of the ganglion, leaving the ophthalmic portion attached to the wall of the cavernous sinus. It was a difficult, dangerous operation, with a very high mortality and great danger of serious complications, especially in the eye, but it generally, not always, put an end to neuralgia for the survivors.

Abbe sought to attain the same result by cutting the second and third branches and inserting a piece of rubber tissue between the ganglion and the foramina. Van Gehuchten, on the ground that regeneration on the central side of a spinal root ganglion or its homologue, the Gasserian ganglion, is impossible, suggested the mere section of the sensory root, allowing the ganglion and its peripheral connections to remain undisturbed. This has the advantage of severing the central connection of the ophthalmic as well as of the other branches. It has been done by Horsley and by Frazier with Spiller's advice. Cushing (8), from an experience of twenty cases, claims that it is not easier to cut the sensory root than to excise the whole of the ganglion, which he has succeeded in doing in all but one or two of his cases, with only one death out of the twenty. There have been some recurrences in Abbe's cases, but, as far as I know, none in cases of section of the sensory root or of complete extirpation of the ganglion.

I therefore would choose one of the latter operations, with a preference for Cushing's, on account of the large number of cases in which it has been successful, with so low a mortality. As to the relative danger of trophic complications in the two operations I know of no facts that would warrant a conclusion.

We may hope that a definite limit has been set to "this hideous disease" by gasserectomy or section of the sensory root, but this is by no means certain. We know that section of posterior spinal roots does not always put an end to tabetic or neuralgic pains in their distribution. True, the recurrences of trigeminal neuralgia have been in cases where the ganglion was probably not completely removed, but in these the recurrent pain has not been limited to the ophthalmic branch. For the past year I have kept an old man free from pain by means of deep injections of alcohol into the second and third branches, whose pain returned in these branches with great severity about a year after a gasserectomy. If it be said that this was an incomplete operation, we have this from Kiliani (20): "Gasserectomy is pronounced the only certain permanent cure by its inventors and their followers. I have treated by injections and freed from pain six cases of intracranial operations, with complete recurrence of pain worse than before the operation. Of these six operations there were four gasserectomies, performed by our best surgeons, and two Abbe operations performed by himself. Therefore, it seems to me there is no permanent cure for neuralgia." We may expect, however, that in the future the cases for which there is neither permanent cure nor long periods of relief will be very rare.

Summary of the Treatment of Trigeminal Neuralgia

1. An attempt to remove all local and constitutional causes, together with incidental relief of pain as far as may be necessary and possible. If no definite, constitutional cause is found, salicylates should be tried.
2. Soothing local measures, especially warmth and protection from irritation. As long as there is a fair prospect of success, these measures should be persevered in without depriving a nerve of its sensibility, for

the pain has some value as an indication that there is a constitutional or local cause to be removed.

3. Galvanic electricity.
4. Analgesics and narcotics.
5. Alcohol injections in the foramina of the face.
6. Deep alcohol injections.
7. All the preceding having failed and the pain being intolerable, gasserectomy or section of the sensory Gasserian root.

CERVICOOCCIPITAL NEURALGIA

Acute neuralgia of the occiput and neck is generally caused by exposure to cold and wet and, as a rule, yields readily to general and local warmth, free elimination, and a salicylate. Phenacetin or a mild opiate, such as codein or Dover's powder, may also be advisable.

Chronic pain in this region should cause a most careful search for organic disease of the bones or spinal membranes. Idiopathic chronic neuralgia is very rare. When it exists the general treatment of trigeminal neuralgia is applicable. Salicylate of sodium and potassium iodid should be given a thorough trial. Galvanism, a large anode on the occiput, with strong currents, if necessary, is especially recommended by Remak. The great occipital nerve can be injected with alcohol where it emerges from its opening in the trapezius and crosses the superior curved line of the occipital bone, 2 cm. from the median line. The branches of the cervical plexus emerging near the middle of the posterior border of the sternomastoid muscle can also be injected, taking care to avoid the external jugular vein, after making it visible by compression at the base of the neck. Painful points along the course of the nerves may also be injected immediately beneath the skin.

BRACHIAL NEURALGIA

Before treating brachial pain as a neuralgia, the possibility of its being a symptom of organic disease of the spine, membranes, cord, or nerve trunks must be carefully considered. If such organic disease is found, the treatment is to be that described under neuritis. Constitutional causes must also be looked for, and, if found, treated, especially rheumatism, gout, diabetes, and anemic or cachectic conditions. Inflammation of the joint or bursal sacs about the shoulder may simulate neuralgia, and yet require altogether different treatment. Oppenheim says: "Taking all in all, I regard a true, pure brachial neuralgia as a rare affection; there is usually a background of hysteria or neurasthenia, of an organic disease, or a constitutional illness (diabetes, etc.)." "I have found more and more that brachial neuralgia is, as a rule, a brachial psychalgia." It is of the utmost importance to recognize the psychic element in such cases, for the right treatment is that of hysteria or psychasthenia, and any other is sure to be unsuccessful. For the neuralgic element proper constitutional treatment is most important. Locally rest, warmth, galvanism, judicious massage, and counterirritation are of use. The nerves should not be injected or stretched as long as there is any possibility of the arm being useful. In amputation neuralgias the nerve trunks may be sacrificed or the spinal roots cut, although even then relief is doubtful.

INTERCOSTAL NEURALGIA

The pains symptomatic of organic disease of the spine, membranes, or cord are not considered here, nor are the referred pains of visceral disease as described by Head. Herpes zoster is treated separately.

The causal and constitutional treatment appropriate to neuralgia in general is indicated here. Gout, syphilis, and states of exhaustion are especially likely to call for treatment. A neurotic factor is often present, requiring psychic treatment. Light blistering over the painful points may help. Local warmth and restriction of the movement of the ribs by a bandage are often serviceable. Stretching of the nerves and resection of ribs have been done, not always with success. It would be better to inject alcohol subcutaneously over the painful points or as close to the nerve trunk as possible.

LUMBOABDOMINAL AND CRURAL NEURALGIA

Pains in the distribution of the lumbar plexus may at first seem to be true neuralgia. Careful examination, however, will almost invariably show that they are symptomatic of disease of the spine or pelvis or of neuritis. In such a case the treatment must be that of the cause, with incidental relief of pain. In the few cases that may be regarded as idiopathic the treatment applicable to neuralgia in general, with local rest, protection, and warmth, is indicated.

PUDENDOHEMORRHOIDAL NEURALGIA

If the paroxysms of pain are in the spermatic cord and testicle a suspensory bandage should be worn and antineuralgic and narcotic drugs used sparingly. A neurotic mental condition is generally present and calls for psychic rather than local treatment. Resection of the testicular nerve has been done by Chipault with success.

Pains in the anus, perineum, and rectum, when not due to organic nervous disease, such as tabes, or to inflammation of the pelvic organs, are generally of an hysterical or neurasthenic nature. Careful search should be made for a local cause, such as hemorrhoids, anal fissure, or urethritis, in order to remove it if possible. If none is found, the pain may be cautiously antagonized by suppositories of opium or cocain, but the general mental and nervous state should receive the chief attention, the treatment being that of hysteria or psychasthenia. Local treatment of any kind, if not really necessary, is often harmful, because it keeps the patient's mind on his pains. A single thorough examination followed by assurance that the local conditions are all right, with concentration of attention on some other condition which can be favorably modified, is often the best treatment.

COCCYGODYNIA

Pain of a neuralgic character referred to the coccyx may be due to local injury, as from parturition or a fall, or to inflammation of the surrounding tissues. A careful examination should be made and any local disease should be treated conservatively on surgical principles. All the cases I have seen have been of an hysterical character, in which treatment of the general nervous and emotional condition was of prime importance, and the less said or thought about the coccyx the better. Excision of the coccyx has generally been a useless and harmful operation performed on a mistaken diagnosis. Unless there is unmistakable visible deformity or gross disease, operation is far more likely to aggravate the complaints than to cure.

HERPES ZOSTER

It is now safe to regard the peculiar eruption of herpes zoster as being in every case the trophic expression of inflammation of the corresponding ganglion of the posterior root, whether spinal or cranial. The older observations of zoster, apparently due to inflammation of the nerve trunks, nerve roots, or posterior columns of the cord without lesion of the root ganglion, were probably wrong because of defective methods of studying the ganglion. In the most typical cases the disease appears to be an idiopathic inflammation of one or more of the root ganglia, analogous to poliomyelitis, due to an unknown specific germ. This form of the disease runs a short course, reaching its height in a few days, and then rapidly subsiding, although some of its bad effects may persist. One attack of this kind confers immunity. Purely toxic conditions may also cause herpes zoster, especially arsenical poisoning. Gout is a possible toxic cause. In addition to the idiopathic and toxic cases, there are symptomatic ones in which the inflammation of the root ganglion is secondary to adjacent organic disease, as in Pott's disease or any of the forms of spinal meningitis.

Treatment as to Cause.—If any primary organic or toxic disease can be discovered, its importance will overshadow that of the complicating zoster, and its treatment will naturally be the first consideration. If the case is idiopathic there is no way at present of influencing its course. Naturally one would keep the patient at rest, open the bowels, and administer salicylate of sodium, salophen, or aspirin. In addition to this, as hexamethylenamin is known to exert some antiseptic effect on the cerebrospinal contents, it would be wise to administer it if the patient is seen early, gm. 0.5 (7½ grains) three or four times daily for an adult.

Local Treatment.—This consists essentially in protecting the vesicles from irritation and infection. Starch or talcum powder with an addition of ten per cent. of the finest powdered boric acid should be applied freely to the affected skin and a binder of fine muslin or linen be smoothly and firmly applied, so as to prevent any friction from the clothing. One to three per cent. of powdered camphor may be added to allay burning and itching. If the pain is severe, instead of the powder a one per cent. ointment of cocain, eucain, or stovain in lanolin and vaselin may be spread on the cloth and applied. A fifty per cent. solution or ointment of ichthyol acts well. [I have had good results by strapping with zinc oxid rubber adhesive plaster when this can be done. But it is necessary to employ a well made and pure oxid of zinc plaster. Even then some patients are found whose skin does not tolerate it.—Editor.] The vesicles should not be opened unless they are so tense as to increase the pain, when they may be incised at the summit. All irritating substances should be scrupulously avoided, for the skin lesions may increase both in extent and in severity after any local application, and then the patient is very likely to think a great mistake has been made, unless he knows that the dressing is of a mild and soothing character.

Late Pain.—After an attack of herpes zoster, it sometimes happens, especially in the debilitated or aged, that the pain persists for weeks or even for an indefinite time. This is probably due to cicatricial changes in the root ganglia. Measures to build up the general health and promote absorption are indicated. Strychnia, iron, and potassium iodid or the iodid of iron are valuable. I see no reason to expect any good from the preparations of phosphorus in such cases. Cod-liver oil may be very useful where it can be digested; if it is not well borne, butter and cream are good substitutes. Small blisters over the affected spinal ganglia may help, and light touches with the Paquelin cautery are still more effectual. Analgesics and narcotics are to be used as in neuritis and neuralgia. The periphery must be protected from irritation of all kinds, mechanical, thermal, and chemical. If the nerve trunk has no important motor functions and other means fail, alcohol injection should be tried. It will not be as effectual as in true neuralgia, because there is organic disease central to the injection, but cutting off impressions from the periphery may do good by giving the inflamed ganglion rest. The epidural injections of Cathelin have a special value in such cases, as the solution can come into close relation with the root ganglia unless they are situated too high. Gowers quotes Sir William Jenner as telling of a patient who, in the days before anesthesia, endured the excision of the skin area affected by zoster, and then, finding he was not relieved, killed himself. Even in a less extreme case than this, other means of relief failing, section of the posterior roots should be tried, although the ultimate value of this operation is still unsettled.

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CHAPTER V

DISEASES OF THE BRAIN COVERINGS

JULIUS GRINKER

INTRODUCTION

Perhaps in no department of medicine is diagnosis more essential for the proper application of treatment than in that of neurology. And, largely because so much time must be devoted to each routine examination, neurologists have earned the unenviable reputation of being only diagnosticians. The author of these chapters has aimed to disprove the charge, not by introducing each disease with an interminable array of details on treatment, but by attempting to supply the reader with a serviceable guide in the management of the most obdurate diseases known to mankind. For reasons that are evident, he has also deemed it expedient to precede the discussion of the treatment of each disease with a concise description of the disease as a whole.

I. DISEASES OF THE DURA MATER

A. PACHYMENINGITIS EXTERNA

Inflammation of the external layer of the dura is usually caused by extension of disease from the cranial bones. Forming the periosteum of the skull, the dura may become infected by mere continuity from fracture or other trauma. By contiguity osteomyelitis, caries, syphilis, erysipelas, or neoplasm of the skull may give rise to dural inflammation.

The *treatment* is purely surgical and identical with that of the underlying conditions.

B. PACHYMENINGITIS INTERNA HÆMORRHAGICA

Of greater importance from a therapeutic point of view is the variety of inflammation involving the inner layer of the dura. It is necessary to review briefly some points in pathology, etiology, and diagnosis.

The essential *pathology* in pachymeningitis interna hæmorrhagica is found on the inner surface of the dura in the form of membranous deposits, in which are imbedded fragile blood vessels with a tendency to repeated hemorrhages. By an increase of fibrinous deposits a mass of variable size may form which resembles a blood tumor, both in appearance and its symptomatology. It is not quite settled whether fibrinous formation precedes hemorrhage or whether the latter is the cause of the former by the formation of organized clots adhering to the dura. Whichever view is correct, the localization of this process takes place with special preference over the parietal and frontal areas in the neighborhood of the longitudinal sinus.

The disease selects individuals past 50 years of age, and especially those who have become weakened by alcohol, syphilis, cachexias of various kinds, or are the subjects of general paresis or other chronic brain disease of the atrophic type. Sufferers from blood diseases, such as pernicious anemia, scurvy, hemophilia, purpura hæmorrhagica, etc., are also prone to develop this condition. In the majority of instances, in addition to the several predisposing causes mentioned, trauma plays the most prominent rôle in the production of symptoms.

Symptoms.—No distinction is made between symptoms arising from hemorrhage and those caused by inflammation. As a rule, the general signs predominate over the focal ones. Depending upon the frequency and amount of hemorrhage, the symptoms will be either mild or severe, with or without remissions of varying duration. In this respect meningeal hemorrhages differ from those occurring as spontaneous intracranial bleedings, in which latter a single large mass of blood is usually thrown into the brain, plowing it up and causing symptoms in one hemisphere, hemiplegic or monoplegic in kind. In pachymeningitis interna hæmorrhagica, however, symptoms are more apt to be bilateral in distribution.

The symptom of greatest importance, because most frequently present, is *headache.* The head pain is not always intense, varies in duration, and is often accompanied by vertigo and a tendency to nausea and vomiting. There are usually general weakness, mental and physical lassitude, and absence of initiative. Of the somatic signs a frequent complaint is the inability to tolerate light and noise—so-called hyperesthesia of the special senses. Insomnia is common. There are, besides, changes in the size of the pupils—they may be small, large, or unequal. Nystagmoid jerkings in the eyeballs and spasmodic twitchings in the muscles of the face or of the extremities are not uncommon. Toward the end there appears somnolence of varying degree; distinct motor paralysis with fever and convulsions may close the scene.

The *prognosis* is exceedingly grave. The tendency is for repeated hemorrhages to occur at short or long intervals, sooner or later leading to a fatal termination.

Prophylaxis

Dural inflammation being always secondary to other disease processes, prophylaxis must confine itself to the treatment of the underlying condition. If the cause be syphilis, a rigid course of antispecific treatment should be instituted. In nephritic and cardiac conditions the heart and kidneys must be treated.

The patient's life must be so regulated as to avoid worry and physical and mental stress. The diet should be nutritious and non-stimulating.

Treatment

To combat the disease itself our efforts are directed toward the prevention of congestion and fluxes to the brain. We endeavor to stop the hemorrhages by the use of ice and cold applications to the head. In robust individuals leeches or wet cups may be placed near the region of the longitudinal sinus, or over the mastoid processes. To reduce congestion we administer laxatives and drastic cathartics, thereby attempting deflection of the blood current from brain to intestines.

Symptomatic Treatment.—For the headache ice to the head and local depletion are indicated. Occasionally the administration of any of the coal-tar derivatives, such as antipyrin, 15 grains (1 gm.); phenacetin, 10 grains (0.6 gm.); aspirin, 10 grains (0.6 gm.), gives prompt relief to the patient. When everything else has failed, and only then, is the hypodermic use of morphin justified. In weak heart with fluttering pulse cardiac stimulants are in order.

Surgical Treatment.—When localizing symptoms, such as one-sided epileptic convulsions, unilateral paralysis, hemianesthesia, hemianopsia, or aphasia, enable one to diagnose the seat of difficulty, an exploratory craniotomy should be made at once.

If successful in finding the nidus of trouble, clots should be carefully removed, bleeding points checked, and the osteoplastic flap replaced. In at least one case has such a procedure saved one of my patients.

A measure advocated by Oppenheim is the combination of bleeding with sweatings. In skilled hands Neisser's brain puncture may not only enable a localizing diagnosis to be made, but may also effect a cure by the removal of large quantities of blood.

C. Traumatic Hematoma

In this connection may be discussed the hemorrhages about the dura which are the result of trauma. These hemorrhages may occur between skull and dura, called extradural, or between the dura and arachnoid membrane, subdural. The extradural hemorrhages are derived from the middle meningeal artery or its branches, and are commonly caused by fracture of the skull. Of all traumatic intracranial hemorrhages those from the meningeal arteries are the most frequent.

The hemorrhages on the inner surface of the dura are derived from torn veins about to empty into the various sinuses, or they may come from the sinuses themselves. In this class belong the meningeal hemorrhages of the newborn, which so frequently terminate in permanent paralysis, idiocy, or epilepsy. As these conditions receive mention in another place, they will not be further discussed at this time. In order to produce a blood tumor, the violence applied to the head need not be excessive. In fact, the skull has often been found uninjured, while extensive bleeding was going on within. In both the extra- and the subdural varieties of traumatic hemorrhage the brain substance is not necessarily damaged, as it is, for instance, in subarachnoid bleeding. Regarding the location, both extra- and subdural hemorrhages take place on the side of the injury, but the opposite side may become implicated by "contre-coup." The amount of blood poured out in each case will largely depend upon the size of the vessel involved. The hemorrhage may become circumscribed and appear in the form of a so-called hematoma, or it may spread diffusely over a large area—in some instances covering almost an entire hemisphere.

Symptoms.—Immediately upon the receipt of an injury, such as a blow upon the head, the patient may suffer from concussion of the brain with its concomitant shock, but soon consciousness is regained. However, as the bleeding proceeds, the patient gradually becomes stuporous and even comatose, unless hemorrhage can be arrested. The interval of time between trauma and the development of symptoms varies from several hours to even weeks. The rule is for a patient to become delirious or somnolent within a few hours. Stupor may then deepen into coma, with slow respiration, retarded pulse, and stertorous breathing—all characteristic signs of cerebral hemorrhage and brain pressure. According to Pagenstecher, the mass of blood must have obtained a circumference of between 37 and 42 centimeters before symptoms of pressure can become manifest. Prior to the development of coma, optic neuritis may be observed on the side of the trauma; also unilateral or bilateral convulsions. On the side opposite to the injury there may appear tonic spasm, opisthotonos, Kernig's sign, and later motor paralysis. As previously intimated, the hemiplegia or monoplegia may be on the same side as the trauma, provided the opposite side of the brain sustained the brunt of the attack by virtue of "contre-coup." Aphasia will indicate that the damage has affected the centers of speech, which are situated on the left side. Hemianesthesia and hemianopsia, if present, will also guide one to a proper localization of the hemorrhage.

In the absence of any focal symptoms it is important to study an existing slight asymmetry of the face, twitchings of muscles, unilateral exaggeration of knee and Achilles reflexes, and to examine for Babinski, Oppenheim, and Gordon signs. The character of the coma may occasionally assist in diagnosis: it is seldom profound and often only transient. A peculiar dazed state following coma is, according to Kocher, quite significant for this condition. If the hemorrhage is at or near the base of the brain, cranial nerve palsies may direct attention to its location. In any hemorrhage of considerable size patients die, unless energetically treated, and even then they cannot always be saved. In slight hemorrhage general symptoms subside and the focal signs resulting from organized clots take their place. Perhaps only then may we discover the lighter grades of hemiplegia, hemianesthesia, and unilateral epilepsy—all of which may become more or less permanent. In some cases the symptoms are so ill defined from the beginning that the condition is not diagnosed until vertigo, headache, weakness, poor memory, and lack of mental concentration become evident, and direct our attention to what had at first appeared as an insignificant trauma.

Diagnosis.—Hemorrhage is occasionally confounded with concussion and contusion of the brain. It is important to remember that in the latter states symptoms develop almost immediately, while in meningeal hemorrhage there is usually a latent interval.

Lumbar puncture may help in the differential diagnosis; if the fluid is yellowish or reddish, it demonstrates the probable existence of hemorrhage. Other tests will corroborate this finding. Even in excessive bleeding the spinal fluid may not differ from the normal. Oppenheim advises cerebral puncture after the method of Neisser-Pollack, not only for diagnosis, but also for treatment.

Treatment

When a hemorrhage from the middle meningeal artery has been diagnosed, the only course open to the physician is to trephine, clear out the clots, look for and ligate the bleeding artery. Before resorting to this radical procedure it is absolutely necessary to have made a focal diagnosis. Timely operation has saved many lives. That non-interference is almost certain to cause a fatal termination has been properly emphasized by W. W. Keen. Wiesmann states that, of 147 cases treated expectantly, 131 died—89.1 per cent.; of 110 cases treated actively, only 36 died—32.7 per cent. In the majority of his fatal cases the clot was not removed because it could not be reached.

Concerning the side of the skull which should be operated on, we must be guided entirely by localizing signs rather than by the site of the injury. Krönlein trephined 4 cases of rupture of the middle meningeal, and in 2 of these he removed the clot, and the patients recovered. In other cases he failed to find it, and those patients died. According to Krönlein, quoted by Keen, in by far the greatest number of cases the clot can be best reached by trephining at a point one inch behind the external angular process of the frontal bone, at the level of the upper border of the orbit. Should this not reach the clot, then a second trephine opening should be made just below the parietal bone at the same level as the former. By not making this last opening he lost the two patients alluded to. By the anterior opening access is had to the main trunk of the anterior branch of the middle meningeal artery; by the posterior, to the posterior branch. In many cases there will be doubt as to which branch is involved; the two openings will, therefore, lead to the desired goal under all circumstances. Having made one or both of these openings, the clots are removed, and either opening may then, if necessary, be enlarged with rongeur forceps, in order to gain access. If the pupils be widely dilated, showing that the clot has extended toward the face, the trephine should be applied about half an inch below the level of the upper border of the orbit rather than at its level.

If no localization signs are present, and the symptoms of hemorrhage proceed, then the life of the patient is in danger, and it will be best to trephine over the place of injury. Several authors, recording cures of meningeal hemorrhage by repeated lumbar punctures, are consequently strong advocates of their use. Dévrain praises this procedure in birth palsies when the fetus is apparently dead. Harvey Cushing, in subdural hemorrhages resulting from prolonged labor, opens the skull and clears out the clots. He maintains that the operation should be done immediately after birth; delay is either fatal to life or else causes irreparable damage to the brain structures, commonly seen as birth palsies of hemiplegic or monoplegic distribution, with or without epilepsy. These views on the surgical treatment are largely taken from Keen, with whom I fully coincide.

II. ACUTE INFLAMMATION OF THE SOFT CEREBRAL MEMBRANES

Introduction.—The leptomeninges may be considered a closed lymph sac, of which the inner layer dips down into all the fissures, becoming intimately connected with the brain substance. From this layer originate the capillary vessels which nourish the brain. Between this and the outer layer we find the so-called subarachnoid fluid. The pia may be considered a serous membrane in the same sense as the peritoneum. Just as infections reach the peritoneum from the viscera to which it is reflected, so do infectious processes spread into the cranium from adjoining territory—the several sinuses, orbit, ear, mouth, antrum of Highmore, etc. Inflammation of the soft membranes is always the result of an infection which has gained access to the arachnoid space, that is, between the two layers of the leptomeninges. The cerebral membranes being continuous with the coverings of the cord and the lining of the ventricular system, we may have an extension of disease from these sources. As there exist no anatomical limitations to the spread of the inflammation, it may become more or less diffuse. This is well exemplified in the variety called epidemic cerebrospinal meningitis, in which the membranes of the brain and spinal cord are involved. The inflammation is not always diffuse. In some cases the convexity suffers most, while in others, as, for instance, the tuberculous variety, the base of the brain is principally affected. In the last location conditions are particularly favorable for the development of inflammation, the large cisterns with their slow lymph currents offering exceptional opportunities for microbes to flourish and to multiply.

By means of a spinal puncture it is possible to diagnose the kind of microbe causing meningitis. In this place mere enumeration of them must suffice. There are:

1. Frenkel's pneumococcus.
2. Diplococcus intracellularis meningitidis (Weichselbaum).
3. Streptococcus.
4. Staphylococcus.
5. Typhoid and paratyphoid bacillus.
6. Colon bacillus.
7. Influenza bacillus.

Cerebral meningitis rarely or never occurs as an independent affection. Nearly all cases originate from an infectious depot outside the cranial cavity. We recognize two paths by which the cerebral membranes may become infected: first, by the blood current, this being the common carrier for the various acute infectious diseases; second, by the lymph current. Through the last route microbes arrive from the contiguous accessory cavities, as the sinuses, the nasopharynx, and the orbit. In like manner inflammations from the cervical membranes spread to the cerebral meninges.

A. Acute (Purulent) Cerebral Meningitis or Leptomeningitis

Symptoms.—This type is usually ushered in with the phenomena of an acute infectious disease, unless masked by the symptoms of another disease of which this is a complication. A typical case commonly begins with chills and fever, severe headache, and cerebral vomiting. Clouding of consciousness soon follows, and in fulminant cases coma may close the scene.

For convenience of description the symptoms are divided into those of *irritation* and those of *paralysis,* although there exists no sharp line of demarcation between them, for irritative often insensibly merge into paralytic phenomena. To the signs of irritation belong the intense headache, insomnia, general hyperesthesia, as well as the hypersensitiveness of the special senses, such as intolerance to light and sound. Other of the irritative symptoms are: nystagmus, twitchings in the muscles of the face and of the extremities, contracted or unequal pupils, spasticity of the muscles of the neck and of the abdomen (boat-shaped abdomen), and rigidity of the back muscles causing arching (opisthotonos). The legs are spastically flexed, and in some cases there may be general convulsions. Kernig's sign is a prominent and characteristic symptom, though not pathognomonic of cerebral meningitis, as was formerly thought.

Toward the end of the disease appear the symptoms of paresis and paralysis. Of the mental functions the psychic reactions are early disordered; increased irritability is present. Somewhat later there is lack of responsiveness—the sensorium becomes clouded up to loss of consciousness. Of motor paralysis we have strabismus, either temporary or permanent. The pupillary narrowing gives way to dilatation, with loss of reaction to light and accommodation. Spasticity in the extremities is replaced by flaccid paralysis, terminating in hemiplegia or monoplegia. In the somatic sphere we may encounter the following symptoms: vomiting, constipation, retention or incontinence of urine. The pulse is relatively retarded early because of vagus irritation, but toward the end it becomes rapid, owing to the paralysis of the vagus nerve.

Diagnosis.—It is not always easy to differentiate the various types of meningitis. For that reason Quincke's lumbar puncture has a diagnostic value of great importance. It will not, therefore, be out of place to describe the technique. Though Quincke originally recommended his lumbar puncture for therapeutic purposes, this method has recently come to be utilized more often in diagnosis. It consists essentially in obtaining a small amount of cerebrospinal fluid by means of a puncture through the spinal membranes, and subjecting the same to a chemical, microscopical, and bacteriological examination.

The technique is as follows: The puncture should be made between the third and fourth lumbar vertebræ in the median line in children, and about one-half inch to one side in adults. This point may be determined by drawing a line from the highest point of the crests of the ilia, which will cross the fourth lumbar interspace. Another aid is the fact that the spinous process of the fifth lumbar vertebra is more prominent than the spinous processes of the sacrum. The patient should preferably be placed on the left side with the thighs flexed upon the abdomen and the head bent forward as far as possible. The point to be punctured should then be marked and the back sterilized in the usual way. The needle, previously sterilized, should be grasped in the right hand, the point placed over the mark on the skin, and held at an angle of forty-five degrees with the surface of the back inclined slightly toward the median line. It should then be pressed forward slowly and steadily. When it has been introduced from two and one-half to three and one-half inches, it may be assumed that it has entered the canal, and the mandril may be withdrawn. When the fluid begins to flow the manometer should be attached and the pressure measured. The fluid is then allowed to flow into a glass tube until it begins to drop slowly, when the needle is withdrawn and the puncture sealed with flexible collodion. If the needle strikes bone it should be withdrawn and reintroduced in a different direction. Should it become clogged, the trocar may be reintroduced to clear it. If the patient becomes dizzy and complains of headache, the operation should be discontinued.

As a rule, no anesthetic is required, but an ethyl chlorid spray may advantageously be used. It is understood that this little operation must be done under perfect asepsis. For diagnostic purposes a small amount of fluid is sufficient.

The normal pressure is, according to Quincke, forty to sixty millimeters of water, though he only considers an increase to over 150 mm. to be pathologic. Under pathologic conditions the pressure may increase to 700 mm. Roughly, we estimate the pressure by the rapidity of the flow, so that, if in a short time thirty to forty c. c. of liquid escapes, we conclude that there is a pathologic increase. The increase of quantity and pressure occurs under various conditions, especially in cerebral tumor, all varieties of meningitis, chlorosis, and edema of the brain.

From the physical appearance of the fluid alone we can often make a correct diagnosis. The fluid is clouded in the purulent and epidemic varieties of meningitis. It may be turbid in tuberculous meningitis, but this is especially characteristic of the different forms of purulent meningitis. If no pus appears to be present, the polynuclear leukocytes will sometimes reveal the purulent condition. In tuberculous meningitis the lymphocytes predominate, while there may be only a few leukocytes present.

The bacteriologic examination is extremely important. In purulent meningitis we find streptococci and staphylococci; in epidemic cerebrospinal meningitis, the Diplococcus intracellularis meningitidis of Weichselbaum; in tuberculous meningitis, tubercle bacilli. The last are usually found in the flocculi; if not present in these, the centrifuge may reveal them. If the fluid appears sterile, as it often does, a culture may give positive results, or the fluid injected into a rabbit or guinea-pig may develop typical tubercles. It will be observed that the careful examination of the spinal fluid may decide an otherwise doubtful diagnosis, although in the majority of instances a lumbar puncture is only confirmatory of the diagnosis.

Lumbar puncture has been tried as a therapeutic measure in most diseases of the brain and cord; it is of great value in relieving pressure when there is an excess of cerebrospinal fluid, and also in removing with the fluid the toxic agents causing the disease. It is, therefore, exceedingly useful in all forms of meningitis, and has been of great service in uremia. Physicians must be warned that lumbar puncture is not a procedure to be used indiscriminately, as a number of deaths have resulted from it in ill-chosen cases. It is contraindicated when arteriosclerosis exists, and in all diseases of the nervous system in which there are not present symptoms of pressure. For diagnostic purposes it should be used only when the diagnosis is really in doubt.

Prognosis.—The prospects for recovery in diffuse acute cerebral meningitis are not good. The majority of cases die after a period of days or weeks. We hear of occasional complete recoveries, but most of these are of doubtful authenticity. Possibly some of these were cases of serous meningitis or of meningeal irritation, so-called meningismus. When acute cerebral meningitis passes on toward recovery, the acute usually changes into a subacute course and the symptoms lose their virulence. Patients recover with intellectual deficit, amounting to idiocy. Deafness from changes in the inner ear is common. Blindness resulting from optic atrophy is one of the sequelæ of leptomeningitis. In the cases of meningitis following other diseases, a suppurating ear, or trauma, such as skull fracture, the mortality is high. The prognosis will depend upon whether surgical intervention is undertaken early or late. Localized forms of meningitis of the purulent variety may recover when the pus depots are thoroughly evacuated.

Prophylaxis

In a disease with so few recoveries prophylactic treatment occupies a prominent rôle. It will be our duty to treat promptly all local suppurating foci near the head, in order to prevent the spread of an infection to the meninges. In all those instances in which a primary disease center is discovered, be it near or far, our duty is to attempt instantaneous clearing out of the same, regardless of its situation. If the starting point of meningitis is in the ear, eye, or intraorbital tissues, these will require a thorough search for suppurating depots. The nose in particular constitutes a prolific source of infection. In every suspicious case of meningitis, therefore, the nose will have to be hunted down, because its lymph vessels have a direct connection with those of the meninges. Its mucous membrane can be treated advantageously in the beginning of every case. Likewise must the frontal and maxillary sinuses receive surgical care. The pharynx being another source of infection, it has to be treated energetically. Any existing tonsillar or pharyngeal inflammation will, of course, receive immediate consideration. For reasons that are evident, abscesses, carbuncles, and other septic foci in the upper portions of the neck may cause meningitis. Erysipelas of the face and head occasionally causes meningitis by disseminating the septic products through the veins and lymph vessels of the scalp. As a matter of course, the underlying conditions will have to be dealt with in no uncertain manner. Even insignificant traumata about the head should be given immediate surgical attention, for from such sources meningitis may develop days and weeks after the trauma had been forgotten and the patient had considered himself well. Caries of the skull bones, osteitis, and intracranial neoplasm, the infectious granulomata, such as gumma and tubercle, may all produce some form of leptomeningitis. The indications for action are clear: extirpate the tumor masses and remove any existing pus from all extra- and intracranial avenues.

Summary.—Whenever there is a sign of beginning cerebral meningitis, the rule is to make a careful local examination of all the cavities and sinuses about the head, then to search out the pus depots, and finally to open widely and drain freely.

Treatment

Specific Remedies.—At present no specific remedy for this disease is known. Mercury has been tried and success has been reported from its use. It was claimed that inunction of mercurial salves into the scalp is capable of saving lives. This appears to me quite doubtful, and I can state that in recent years not much is heard of mercurial treatment.

[Unguentum Credé should be tried in every case. It should be employed in large doses, one-third of a pot three times daily. A number of cases have recovered after its employment.—Editor.]

Symptomatic Treatment.—In the initial stage the fever should be controlled by bathing. As in other conditions we are occasionally obliged to make use of the salicylates, also of antipyrin, acetanilid, and phenacetin. Intense headache may be relieved by applications of cold to the head, the administration of bromids, withdrawal of blood by means of leeches applied to the mastoid processes, the temples, angles of the orbit, and the nose. To most patients an ice bag applied to the shaved scalp will be agreeable. If this cannot be well borne, cloths wrung out in cold water and applied to the head and neck will give relief for the nervous irritability and the fever. Laxatives are indicated, because of the prevailing tendency to constipation throughout the course of this disease. The kidneys, too, must not be forgotten, as retention may occur. The patient should be at absolute rest in a cool, darkened room. All sources of irritation from without, such as noises, bright lights, etc., should be avoided. In recent years draining of the ventricles into the subdural space by lumbar puncture, frequently repeated, has found many advocates. In children, when the fontanelles are still open, they are utilized for the same purpose. The benefits are obvious in some cases, but many patients are not at all influenced by any mode of treatment.

B. Epidemic Cerebrospinal Meningitis

See Vol. II, Section I, Chapter XII.

C. Tuberculous Meningitis

This type of meningitis occurs most frequently in childhood; the atypical forms are not infrequently seen among adults. The age from two to twelve years seems to be most favorable for its development. A superficial glance at the pathological anatomy of this fatal disease will explain most of the symptoms. It is well to bear in mind that the pathological processes take place principally at the base of the brain, therefore it is also called basilar meningitis.

The tubercles, which are no larger than a millet seed, follow the course of the blood vessels at the base of the brain pretty closely. The circle of Willis, the Sylvian fissure, the surface of the pons, the lower aspect and sides of the cerebellum are the parts most often involved. The tubercles are often found over the bases of the central convolutions and it is important clinically to remember that the facial twitchings observed in this disease might be produced by this distribution. Postmortem the great lymph sacs at the base of the brain seem to be distended with serofibrinous effusion, so that the arachnoid is forced up, is stretched, and has a milky gray color. Numbers of tubercles can be seen protruding through this milky and opaque superstructure.

The cranial nerves are often found imbedded in the exudate. The superficial layers of the brain may likewise be studded with tubercles, and we may have in addition a meningoencephalitis.

Through the tela, which carry the choroid plexuses into the interior of the brain, we have extension of the disease, in consequence of which acute hydrocephalus and symptoms of pressure follow.

Symptoms.—All the manifestations of tuberculous meningitis may be classified under general symptoms, mostly caused by pressure, and focal symptoms, caused by direct implication of nerve tissue.

Among the *general* symptoms are: headache, vertigo, vomiting, slow pulse, disturbance of the respiratory rhythm, jactitations, convulsions, hyperesthesia, delirium, coma, involuntary sphincter action. On the border line between general and focal symptoms are: spasticity of the extremities and of the masticatory muscles, grinding of the teeth, rigidity of the neck, and probably the transient localized twitchings.

Headache is a very constant symptom and may be caused either by direct pressure of the exudate upon the nerve filaments of the dura, or else indirectly by the fluid from within the ventricles. The fever in tuberculous meningitis is never high, except in the terminal stage, when it may run up to 104-106° F. Its usual range is from 100.5° to 102.5° F.

The pulse at first may be retarded, owing to stimulation of the vagus center in the medulla. Late in the disease, when paralysis of the vagus center begins, the pulse becomes rapid and irregular.

Respiration becomes arhythmical early in the disease and is the result of a moderate amount of brain pressure; when the pressure rises, and when coma has supervened, respiration is deep and slow. When the pressure has become very high, and if the retarded or vagus pulse comes on, respiration becomes irregular and at times ceases altogether. When in the further course of the disease vagus paralysis manifests itself by the acceleration of the pulse, the respirations become very labored (Cheyne-Stokes), and are finally extinguished.

The clouding of the sensorium is probably due to pressure, as well as the vertigo and vomiting occurring upon postural changes. Likewise must optic neuritis—better called papilledema, according to Cushing, of Baltimore, and Sänger, of Hamburg—be ascribed to increased brain pressure and edema of the nerve-sheath. Possibly also the pupillary anomalies may be produced by the same cause exerted on the cortical or nuclear centers; pupillary inequalities indicate unequal involvement of the two sides.

Meningeal thickening is probably responsible for the various muscular rigidities which cause, in the neck, trismus and opisthotonos, in the trunk, arching and boat-shaped abdomen. The various hyperesthesias of skin and special senses may be similarly explained. The tendon reflexes are exaggerated early in the disease, later they are either reduced or entirely absent. The Kernig sign is often present on one or both sides, but only its presence is of diagnostic value.

Vasomotor disturbances are common, so that a light stroke over the forehead or chest may leave a red streak for some time. This is the so-called *tâche cérébrale* of Trousseau, which was formerly regarded as pathognomonic of tuberculous meningitis, but we now know that it occurs in other nervous disorders.

Constipation is, as a rule, noticed early; later in the course of the disease there are involuntary discharges. The urine may contain small amounts of albumin, sometimes a slight amount of sugar.

Great emaciation is a well-marked feature in tuberculous meningitis, so that in a few days the patient may be reduced to a mere skeleton.

Focal symptoms are caused by the accumulation of tubercles in certain cortical areas with subsequent destruction of brain substance and by direct involvement of cranial nerves. Thrombosis of the Sylvian artery may cause softening of the brain and hemiplegia may result. The most important focal symptoms are the paralyses of cranial nerves, especially of the third (strabismus, pupillary differences, myosis, or mydriasis). Hemiplegias, preceded by symptoms of irritation, such as localized twitchings, are common. Aphasia, particularly of the motor type, is occasionally met with, and may be due either to blocking of the left Sylvian artery or to a tuberculous deposit in the neighborhood of Broca's convolution. Tubercles in the choroid, when found, are of greatest diagnostic import. Optic nerve involvement has already been mentioned.

Diagnosis.—A typical case of tuberculous meningitis rarely, if ever, offers difficulties in diagnosis. It is the atypical cases that tax the physician's diagnostic skill. A correct diagnosis can be made even in the absence of some of the characteristic symptoms, if one remembers the particular grouping of symptoms in this disease: the gradual onset; the irregular type of fever; the peculiar quality of pulse—irregular at first, then somewhat retarded, and accelerated toward the last; the violent headache; the ocular and facial paralysis; the pupillary differences; the somnolence, convulsions, and coma. All these constitute a symptom group that makes its own diagnosis.

However, there are atypical forms of tuberculous meningitis, particularly in adults, in which diagnosis is almost impossible. For instance, the disease may remain latent or be entirely overshadowed by the primary affection. In some cases there is complete absence of fever or even a subnormal temperature. Delirium tremens has been known to mask the disease; and focal symptoms, such as monoplegia, hemiplegia, aphasia, Jacksonian fits, have occupied the foreground. The last varieties are probably cases of localized tuberculous meningitis that become generalized later. Then there is a form of disease which, under certain circumstances, can be easily mistaken for brain tumor. Of course, this is not likely to happen in the ordinary type of tuberculous meningitis with acute hydrocephalus, which runs a course of from four to six weeks. But there is a chronic form of the disease in which the differential diagnosis from tuberculous tumor is almost impossible, on account of the similarity in symptoms. Allen Starr mentions the following points as of some value: Headache is more severe in meningitis and more continuous; there is greater hypersensitiveness to light, sound, or touch in meningitis; and optic neuritis develops less frequently, less rapidly, and with less intensity than in tumor. Tubercles upon the choroid are found more frequently in meningitis than in tuberculous tumor.

Of all infectious diseases, none has probably more often been mistaken for meningitis than typhoid fever. More than once have I been called to see a case of supposed tuberculous meningitis in which the usual fatal prognosis was given, and which turned out to be a case of typhoid fever with recovery. The symptoms in some cases may be so similar—even aphasia and hemiplegia have been observed in typhoid—that only a positive Widal reaction and a lumbar puncture may definitely clear up the diagnosis.

Certain febrile digestive disorders in children may resemble meningitis in that the patients suffer from headache and pains in the muscles, vomiting, and constipation. There are anorexia, coated tongue, abdominal pain, and sometimes decided photophobia. In addition there may be an irregular fever, ranging from 99.5° to 102.5° F.; the pulse may be accelerated, but regular. Although these patients appear moody, irritable, semistuporous—all symptoms found in the prodromal stage of tuberculous meningitis—yet careful inquiry will almost always elicit the fact that there has been some indiscretion in diet with a rather sudden onset of symptoms. The further course of the disease will invariably settle the diagnosis.

Tuberculous must also be differentiated from acute purulent leptomeningitis. In the latter the characteristic prodromata are lacking; further, it has a sudden onset and a rapid progress; the temperature runs higher and vacillations are infrequent. Cranial nerve involvement is more pronounced in basal than in meningitis of the convexity. Besides, cerebrospinal meningitis, as opposed to the tuberculous variety, has a rapid onset with high fever, and spinal symptoms are present. In the epidemic disease there are early rigidity of muscles, opisthotonos, tremor, jactitation, and often herpes.

After everything has been said, differentiation of one type from another, in some instances, is almost impossible. For this the careful examination of the cerebrospinal fluid will be helpful. It is characteristic, though not pathognomonic, for tuberculous meningitis that the fluid on standing precipitates in the form of small coagula, which adhere to the walls of the tube. When loosened, the coagula separate into flocculi. The tubercle bacilli, for which careful hunt must be made, will usually be found in these fibrin masses. An additional point to remember is that in a cellular count the mononuclear lymphocytes preponderate, very few polynuclears being present.

Treatment and Prophylaxis

Little can be said regarding any specific treatment of this fatal malady. We have no such serum as has reduced the frightful mortality of epidemic cerebrospinal meningitis. The fatal character of the tuberculous variety of meningitis was recognized ever since the disease has been known. In recent years several cures have been reported. These cures, if they are genuine, did not result from any specific treatment used, but occurred under symptomatic medication. Not being, then, in the position of being able to effect a cure, we shall at least make efforts to prevent the development of the disease, if that is possible.

Prophylaxis.—As in other forms of tuberculosis, two factors are necessary to produce the disease, namely, (1) a hereditary predisposition to tuberculous disease, and, what is even more important, (2) a source of infection. It will be our aim to so regulate the patient's life as to prevent or make it difficult for tubercle bacilli to find lodgment in his economy. A child known to be predisposed to tuberculosis should not be brought in intimate contact with those suffering from the disease. It must be kept out of doors most of the time during the day. Windows of bedrooms should be kept open day and night. When the child is old enough to attend school it must be carefully guarded against too strenuous school tasks. Strict attention should be paid to school hygiene in every detail. If the public school does not comply with the hygienic requirements, the child should be sent to a well-regulated private school.

In addition there is to be observed *local prophylaxis,* which means the prevention of the spread of disease from sources on the patient's own body, by so-called autoinfection. Wherever tuberculous glands are discovered on the patient, they must be promptly dealt with both medically and surgically. Any existing tuberculous disease of the nose or pharynx requires constant attention so as to prevent its spread to the cerebral membranes. Another prolific source for the dissemination of tuberculosis is the osseous system. Tuberculous caries and periostitis should be treated according to the best rules of our art.

Treatment of Symptoms.—In the fully developed disease we treat symptoms as they arise. The treatment does not differ from that already outlined for the other varieties of meningitis. In the presence of this disease we are powerless to effect cures, but we may relieve symptoms. A few years ago the administration of iodoform, so helpful in other forms of tuberculosis, was tried, but without success. When the tuberculin era began, much was hoped from tuberculin injections. Our experience has been that not only were patients not benefited by its use, but some even became worse as a consequence. At present only few physicians continue to use tuberculin in this affection.

Operative Treatment.—By reducing the pressure in the cerebrospinal fluid it was thought that symptoms might be relieved and time gained for the disease processes to become regressive. For this purpose the ventricles are being drained by means of brain puncture. The effects obtained are transient in character and discourage a continuance of this procedure. *Lumbar puncture* enjoys greater popularity. Fürbinger used lumbar puncture in 37 cases, but the results were unsatisfactory; none of his cases showed permanent improvement. Neither can Heubner boast of lasting favorable results, but he saw temporary improvement in some of his cases, especially as regards the relief of pain. Freyhan reports that, after the removal of 60 c. c. of cloudy serous exudate, one of his patients began to convalesce, and in three weeks more he left his bed well. In this case the cerebrospinal fluid showed tubercle bacilli. Henckel published a similar case. Riebold achieved a complete cure in one of his cases by daily lumbar punctures. The fluid contained numerous tubercle bacilli and inoculation tests were positive. Rieken has recently tried lumbar puncture in six cases from Quincke's clinic; all died. There are still other reports of cures by this method. It must be admitted, then, that lumbar puncture often relieves symptoms, and that exceptionally tuberculous meningitis may be cured by it.

[It is the method of treatment which should be employed in every case. Aside from the possibility of cure, the symptomatic relief given is so great that the whole clinical picture is changed by it. While I have never been so fortunate as to have any one of my cases recover, the absence of pressure and toxic symptoms has done away with much suffering, both to the patient as well as to the family. The duration of the disease, it has seemed to me, has been prolonged by this treatment.—Editor.]

D. Chronic Cerebral Meningitis

From the clinician's point of view this disease is of little importance. The majority of cases occur in connection with brain syphilis. The acute and subacute cases of cerebral meningitis occasionally merge into the chronic type. Chronic meningitis occurring in an individual who has had syphilis, especially if mental symptoms are prominent, means the coming of general paresis. In connection with active syphilis, chronic cerebral meningitis may cause optic atrophy terminating in blindness. In the large majority of cases chronic meningitis is caused by luetic infection, chronic alcoholism, or general paresis. In frankly syphilitic cases the disease limits itself to the base of the brain, while in the alcoholic and paretic varieties the convexity is principally involved. Rarely internal hydrocephalus terminates in chronic meningitis; then the cerebellar cortex and the ependyma of the ventricles may be the seat of the disease.

Treatment

This will be entirely etiologic. If syphilis be the etiologic factor, energetic treatment with mercury and iodids is indicated. In general paresis antispecific treatment is of little or no avail. However, patients should receive the benefit of this treatment, as it is not always possible to determine the degree of active syphilis still present in cases presenting the clinical signs of general paresis. In chronic meningitis the result of an acute type in which hydrocephalus is present lumbar puncture or ventricular tapping may be tried. Cures have been reported from such treatment.

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CHAPTER VI

CIRCULATORY DISORDERS OF THE BRAIN

JULIUS GRINKER

1. CEREBRAL ANEMIA

Introduction.—Formerly much space was given to descriptions of anemia and hyperemia of the brain. Both of these circulatory disorders were discussed as morbid entities; their etiology, symptomatology, and pathology received lengthy mention, but very little of value was said under the heading of treatment. At the present time cerebral anemia and hyperemia are spoken of as symptoms of functional or organic disease; consequently they receive but scant consideration in the modern textbook. From the point of view of therapy, however, these conditions merit more than passing mention. Much can be done to ward off a serious attack of brain disease if the preceding vascular symptoms can be recognized and treated at an early stage.

Cerebral anemia is often only part of a general anemia. Quite frequently it is produced by a feeble condition of the heart, causing so-called syncope. In the brain itself it is possible to have a localized anemia from neoplasm, vascular thrombosis, or hemorrhage; hydrocephalus may also produce it by pressure upon the surrounding tissues. We distinguish between total and partial, acute and chronic forms of anemia.

Symptoms.—(a) In the *acute* variety of cerebral anemia we have the ordinary symptoms of syncope. The patient experiences a drowsy feeling and falls into a "faint." There appear dimness of sight, ringing in the ears, and inability to remain standing. In addition there are noticed pallor of the face, coldness of the extremities, sighing respiration, and feeble heart's action. If this state continues, consciousness may be lost and general convulsions appear, possibly ending in death.

(b) In the *chronic* variety the patient experiences subjective sensations of tingling and numbness in the extremities and a peculiar dull pressure-headache. Black spots may be seen before the eyes, or there may be heard noises and ringing in the ears; giddiness is more or less constant. Physical and mental weakness may be so marked that even to speak is a great effort. In the severer grades of anemia a somnolent or stuporous state develops, with delirium, picking at the bedclothes, and inability to sit or stand. The special senses may be functionally impaired, so that the patient cannot see or hear distinctly.

Prognosis.—The prognosis varies according to the underlying conditions and the patient's constitution. It would appear that the more rapidly symptoms develop the worse is the outlook for recovery.

Treatment

Prophylactic treatment is directed against the various causes of general and local anemia, which is discussed under a separate heading.

Treatment of the Acute Attack.—To combat this no time should be lost by the attending physician. The objective point is to cause immediate improvement of the cerebral circulation. It is absolutely essential to have the patient placed in a horizontal position, in order to determine a better flow of blood to the brain. In some cases it is best to raise the lower extremities and to depress the head, in order to permit the bloodless brain to receive an adequate supply of blood. When necessary, this position may be maintained for days and weeks. Tight bandaging of the extremities, for the purpose of emptying the blood and forcing it into the brain, has been tried with success. All mechanical obstruction about the patient's body, such as close-fitting dresses, corsets, etc., should be removed or loosened, in order to stop interference with the proper circulation toward the brain. At the same time it becomes necessary to cause stimulation of the cardiac and respiratory functions.

Externally, cold water is to be dashed upon the face and the body flapped with towels dipped in cold water; mustard plasters are applied to the region of the heart. Cold can be alternated with hot towels and placed over the body, leaving out the head. Sinapisms may be put upon various portions of the chest and back, shifting from one spot to another. Ammonia, or spirits of camphor, may be advantageously applied to the nostrils. In cases showing a tendency to persist we stimulate the skin by means of the faradic brush and apply the electric current to the sides of the chest and over the region of the heart.

Internally coffee, alcohol, or ether are of decided benefit. Champagne is an efficient remedy. Repeated hypodermic injections of ether in doses varying from 10 to 30 drops have been found efficacious in reviving a flagging heart. Oil of camphor has been used in the same manner and for the same purpose. Hoffman's anodyne in teaspoonful doses and 10 to 20 drops of ether on a lump of sugar are some of the other remedies advised. When life appears to be threatened in either acute or chronic cerebral anemia, we do not hesitate to resort to transfusion of normal salt solution, as by this means many patients have been saved from almost certain death.

For the obstinate sleeplessness of chronic cerebral anemia nothing equals in efficacy the imbibition of a night draft, consisting of either a glass of beer, wine, or even whiskey in small quantities. With this may be combined the postural treatment, viz., horizontal position of head and elevation of feet. In many cases this alone is sufficient to produce sleep. The administration of bromids in anemic conditions of the brain is to be deprecated, for this is certain to aggravate symptoms.

An acute attack of cerebral anemia being frequently only a symptom of some chronic disorder, it becomes imperative to search for the cause and, when found, to apply the appropriate remedy. If cardiac conditions are found to be the underlying etiologic factor, treatment will be directed to the heart. When the lungs are at fault they must receive treatment. A blood examination is to be made in every instance. Each organ should be investigated in its turn and treated according to the rules discussed in other portions of this work.

The nervousness often accompanying cerebral anemia requires perfect rest in bed, good food, and ferruginous tonics. The Weir Mitchell rest cure in all its details will here find a most fertile field for useful application.

2. HYPEREMIA OF THE BRAIN

Introduction.—Like cerebral anemia, this condition is not a disease entity, but constitutes an important symptom in several functional and organic diseases. The early recognition and treatment of it, however, may delay the advent of serious organic disease, of which it may be a remote or immediate warning.

We distinguish between active and passive hyperemia of the brain; also between general and partial, acute and chronic cases.

Active Cerebral Hyperemia.—Some deny that this type of hyperemia is ever a distinct pathological condition, maintaining that a certain degree of active hyperemia is physiological during mental labor, and that it is difficult to draw a line between the pathologic and the physiologic. Most writers, however, are of the opinion that there is a pathological type of active hyperemia, which is induced by mental exertion or emotional stress. Excessive cardiac activity with or without hypertrophy of the heart is a common cause. Fluxes to the brain by insolation, infection, intoxication—especially alcoholic—tea, coffee, and other stimulating beverages, are capable of producing active hyperemia in the brain. Cerebral inflammation and tumors are often accompanied by local hyperemia in their vicinity.

Passive Hyperemia.—Passive hyperemia can be caused by anything which prevents the flow of blood to the brain. The obstruction may be central, such as cardiac lesions at the mitral or tricuspid valves are capable of producing, or there may be a hindrance in the pulmonary circulation. Passive congestion of the brain may also be caused by tumors or enlarged glands pressing upon the veins of the neck or in the axilla, in this manner preventing the return flow to the heart. Among the intracranial causes of this condition must be mentioned tumors of the brain so situated as to press upon the vascular structures, particularly the vena magna, or veins of Galen.

Symptoms.—Both active and passive hyperemia of the brain are characterized by a feeling of increased pressure and heaviness in the head, vertigo, and cephalalgia. In the lighter grades of active hyperemia there is a sensation of heat and fulness in the head, which often prevents sleep. In addition the eyes are blurred; there is an inability to see objects distinctly, or there may be ringing in the ears; also pulsation of the cerebral arteries with thumping headache. In the severer grades of this affection there may be stupor, cloudiness of the sensorium, confusion of the mind, and peculiar twitchings. In these cases convulsions, transient paralysis, and mild aphasia are not rare.

In the passive variety of cerebral hyperemia symptoms of heaviness, somnolence, and depression prevail.

Prognosis.—The prognosis of cerebral hyperemia depends upon the cause. The condition itself usually passes off, except when it occurs as an initial warning of cerebral hemorrhage, in which case it is followed by an attack of apoplexy.

Prophylaxis

Hygienic measures for the prevention of cerebral hyperemia include the avoidance of mental and emotional stress. Individuals with atheromatous arteries and those suffering from syphilis are particularly warned to avoid pursuits in which the element of hazard subjects them to occasional "nerve storms," as these may first cause hyperemia and later hemorrhage from vessels of the brain. For similar reasons excessive study and games of chance are unsuitable for such individuals. Stuffy, overheated rooms should not be frequented, and in consequence theaters and parties are better left alone. The hygiene of occupation requires cool, airy rooms to work in; that of recreation demands that mental labor should be alternated with muscular exercise in the open air. Swedish movements are excellent for this class of patients. Of outdoor sports horseback riding has many advocates, as it has a tendency to draw the blood from the brain to lower parts and to give the rider a feeling of exhilaration. It has also been found very efficacious against insomnia, a troublesome symptom in cerebral hyperemia. Brain workers who suffer from abnormal fluxes of blood to the cerebrum must be persuaded to take up some form of manual labor; best of all farming.

Patients must not partake of large meals. The food must be digestible and assimilable; small meals frequently taken are preferable to the three-meal system. The ideal diet, if it can be carried out successfully, should be vegetarian. Midnight suppers are to be banished. No alcoholic drinks are permitted, especially in the evening, before bed-time. Coffee and tea are to be given over to those differently constituted. Tobacco, except in very small quantities, acts injuriously by promoting cerebral hyperemia through its effects upon the heart. In brief, prophylaxis against cerebral hyperemia consists in a well-regulated life with good habits, a moderate amount of mental labor, with some outdoor exercise. These rules apply equally to those cases which are a result of, or accompanied by, nervous or visceral organic disease.

As passive cerebral hyperemia is not an independent affection, but nearly always the result of disease elsewhere, such as heart and lungs, these organs must be treated. In such cases it may be necessary to use digitalis, strophanthus, or strychnia, if the heart be the organ at fault.

[Arteriosclerosis should be looked for in all of these circulatory cases. In my experience this is the cause in the greater number of patients. Certainly more relief is given, and more patients are cured, by the treatment of arteriosclerosis than by any other mode of treatment.—Editor.]

In respiratory affections with frequent cough small doses of opium may be prescribed, in order to prevent a damming back of blood into the brain. If the congestion is caused by glands or tumors pressing upon the veins of the neck, they should receive surgical attention.

Treatment

An attack of *acute cerebral hyperemia* requires immediate energetic action; delay may mean the development of cerebral apoplexy. The patient must be placed in a cool, dark room, with the head elevated, in order to prevent the flow of blood to the brain and to divert the blood current to the extremities. All tight clothing about the body should be loosened to permit free respiration, thus accelerating the flow of blood to the heart.

An ice cap should be placed upon the head after the hair has been thoroughly moistened with cold water. Cold ablutions to the head and spine are also useful. Physicians do not, as a rule, recommend *general* cold baths, but there is no more powerful remedy, when properly used, to determine a flow of blood through the skin and away from the brain. It must always be remembered that before and after the bath ice or ice-cold applications should be applied to the head. In old people with atheromatous arteries cold baths are not to be used; neither can very hot baths be recommended. In mild cases of cerebral hyperemia cold footbaths have given immediate relief. The feet are allowed to remain in the water ten or fifteen minutes, or until the reaction occurs, which is a reflex contraction of the blood vessels in the brain. Some advise the addition of mustard flour to each bath, others order the legs and feet to be kneaded and massaged while the patient remains in the bath.

Withdrawal of Blood.—Direct withdrawal of blood will effect a rapid reduction of the entire volume of blood in the brain. Local blood-letting, leeches (in small children one to two, in adults six to eight), cupping, wet or dry, occasionally act efficaciously. Blood may be taken from the back of the neck at the junction of hair and skin, from the region of the mastoid processes, the temples, and inner angles of the eyes, and also opposite the longitudinal sinus.

In a plethoric individual with symptoms of threatening hemorrhage immediate withdrawal of large quantities of blood may prevent the attack. Before resorting to blood-letting, however, one must ascertain that the heart is powerful and the pulse tense and full. General blood-letting is contraindicated in cases with pale face, small and feeble pulse, irregular heart action, and particularly in the anemic and the senile with atheromatous arteries. We are to be especially careful in the last type of patient, as he is prone to develop thrombosis, and we may hasten this process by bleeding. In addition to direct withdrawal of blood by venesection or cupping, we aim to deflect the blood current from brain to distal parts of the body by other means. For this the various laxatives are utilized, particularly the salines, such as Rochelle salts, Hunyadi water, and Carlsbad salts. Of cathartics croton oil takes the lead. The dose is 2 drops, made into an emulsion, and repeated every half hour until purgation occurs. Infusion of senna leaves, three to four tablespoonfuls every two hours, until effects are procured, is a favorite remedy with some. Others prescribe jalap, rhubarb, and calomel in combination, or calomel alone in 5-grain doses, followed by salines; also castor oil, in one to two-tablespoonful doses.

Diaphoretics are sometimes beneficial. Antipyrin, 5 to 10 grains (0.3-0.6 gm.) three times daily; phenacetin, in 5 to 10-grain doses (0.3-0.6 gm.), and the salicylates, may be given alternately, for these remedies deplete the circulation by causing perspiration and reduction of pressure in the arteries. Patients must be warned to partake of fluids but sparingly, as any extra amount of liquid increases the heightened arterial tension.

Symptomatic Treatment.—In congestion of the brain, accompanied by restlessness, convulsions, delirium, or spasm, the administration of nerve sedatives is indicated. They are not to be used in those depressed patients who are somnolent and threatened with coma, nor in those with weak heart and a rapid pulse. Sedatives can be given by mouth; in some instances morphium, gr. 1/10 to 1/5 (0.006-0.012 gm.), hypodermically, is to be preferred. In cases of vomiting, restlessness, and delirium, it is better to give chloral hydrate per rectum, in doses not exceeding 30 grains (2 gm.) every three hours, until relief is obtained. When there is danger of collapse, stimulants should be administered with a free hand. In severe grades of hyperemia treatment must be directed toward the prevention of paralysis of the respiratory and cardiac centers. The heart must be stimulated with injections of camphor, strychnin, alcohol, ether, or musk. Externally sinapisms, turpentine, and hot applications are used to the lower extremities, while the head is kept cool by ice. It is in these severe cases that local and general blood-letting save lives.

Treatment of Chronic Hyperemia.—Chronic hyperemia is treated upon an etiologic basis. Proper hygiene and the avoidance of mental stress and alcohol in any form must be insisted on. A wholesome, non-stimulating diet and moderate outdoor exercise are other prophylactic requirements. The indications here are to so regulate one's life as to prevent constant overfilling of the cerebral vessels. Only exceptionally is blood-letting necessary. It is here that horseback riding and Swedish gymnastics celebrate their greatest triumphs. The diet should consist of easily assimilable and digestible food, preferably vegetables, fruits, such as pears, prunes, and apples. Meat and alcoholic stimulants are natural enemies of the chronically hyperemic. Turkish, Roman, and other hot baths are not to be indulged in by this class of patients. There can be no objection to the use of cold footbaths, or the so-called sitz bath. Daily cold ablutions to spine and head are recommended. The temperature of the bath must be adapted to the age, strength, and the reactive abilities of each individual. The surest proof that the bath is beneficial is when the patient feels refreshed after it and can readily get warm; otherwise it does harm.

One of the most difficult problems in chronic cerebral hyperemia is how to overcome the insomnia. This symptom, to be properly treated, requires a thorough examination of all etiologic factors. The patient's habits, diet, occupation, time of work, sleep, state of strength, will all have to be scrutinized with a view to correct faults. The main point in the treatment is strict regulation of the patient's mode of life, the removal of everything that may banish sleep, the reduction of mental labor, and the increase of physical exercise appropriate to the individual's strength. In acute cases of insomnia we must not despair, for we still have at our command the various hypnotics, of which sulphonal, trional, veronal, are only a few. In exceptional cases, and only occasionally, morphin in a single large dose may be administered. Under no circumstances must any of these drugs be left to the indiscriminate use of the patient himself.

In chronic cases of insomnia sleep-producing remedies are better avoided altogether. On the other hand, bromid of sodium or potassium in moderate doses, say 15 grains (1 gm.) three times daily, is highly recommended by Hammond and others.

[A reliable preparation of ergot combined with the bromids has been very serviceable to me in these cases. For many years I have used this combination in all forms of cerebral hyperemia for symptomatic indications. It may be given for a long time, but when evidences of bromism develop the ergot can be continued without the bromids. In many instances ergot alone is beneficial.—Editor.]

An excellent remedy is rubbing the spine and head with a turkish towel dipped in ice-cold water once or twice during the evening, or even at night. Sufferers from insomnia should not work after 6 or 7 in the evening, should eat small meals at night, must take walks in the fresh air, avoid sleeping in the daytime, rise early, and discard coffee, tea, and liquor.

The treatment as outlined is applicable also to cases of cerebral hyperemia occurring with organic intracranial disease.

3. CEREBRAL HEMORRHAGE

Hemorrhage may occur in the brain membranes or in the brain substance itself. Clinically the various kinds of hemorrhage in the cranial cavity cannot always be differentiated. From a therapeutic point of view all varieties may be included in one large group, as the treatment is practically the same regardless of the location of the hemorrhage.

Etiology.—Two factors are required to produce cerebral hemorrhage: a diseased artery and an increase in blood pressure. The hypertension in the arteries is often associated with chronic interstitial nephritis. The vessels are atheromatous, and miliary aneurysms have been found on them. It is by the giving way of an artery whose walls had been weakened by disease that hemorrhage is possible. In the presence of congestion, or increased blood pressure, in diseased arteries, we have hemorrhages, either large or small, depending upon the caliber of the vessel.

There is a type of hemorrhage which occurs during or after birth, which is commonly due to the mechanical force applied to the head, either by a narrow parturient canal or by the forceps. These hemorrhages are mostly meningeal and bilateral. Some of the infectious diseases of childhood may give rise to hemorrhage, particularly when there is sinus thrombosis present. Charcot and Bouchard, in 1864, first described the small aneurysmal dilatations frequently seen in those arteries which have given way in hemorrhage, and the majority of physicians still believe that the weakening of the vessel wall is the principal cause of hemorrhage in the brain. As the ganglionic arteries from the circle of Willis are oftenest the seat of this pathological change, hemorrhage occurs in them with great frequency. Of these the most common seat of hemorrhage is in the small artery supplying the lenticular nucleus, which Charcot named the artery of hemorrhage, otherwise called the lenticulo-striate artery. Hemorrhages may also occur in the region of the pons, cerebellum, corpora quadrigemina, and medulla.

The age of hemorrhage is between 45 and 65, the very time of existence when, by chronic alcoholism or repeated physical and mental stress, the arteries begin to degenerate.

Although thrombosis is the more usual lesion, yet syphilis is a factor in about one-third of all cases of hemorrhage. Hemorrhage in the brain is not uncommon in purpura hæmorrhagica, pernicious anemia, and the various hemorrhagic diatheses.

In a case predisposed to hemorrhage the attack itself is often caused by coughing, sneezing, lifting of heavy weights, straining at stool, coitus, or by severe emotional disturbances, such as fright and anger. Even intense joy has been known to bring about an attack.

Symptoms.—One may speak of headache and giddiness as premonitory symptoms of hemorrhage, if they occur in those whose arteries are likely to rupture. Even ptosis and transient diplopia have been observed to precede an attack of cerebral hemorrhage. In many cases the symptoms of cerebral congestion may have been present, but failed to attract attention. Hemorrhage of the brain usually begins suddenly, with a so-called "stroke," a name formerly applied exclusively to this vascular affection.

The symptoms are divided into (a) *general,* common to all hemorrhages of a certain size irrespective of situation; (b) *local* symptoms, which indicate their position.

(a) The principal *general* symptoms are: sudden loss of consciousness, varying from slight confusion to deep coma; stertorous breathing, which may be of the Cheyne-Stokes type; full pulse, subnormal temperature, and loss of control over the sphincters.

(b) The *local* symptoms will vary with the position of the hemorrhage. As previously stated, the branches of the middle cerebral artery are particularly prone to rupture, and of these the lenticulo-striate is especially liable. Hemorrhage from this small artery, which supplies the lenticular nucleus having the motor tract on its inner side, will give rise to symptoms of paralysis on the opposite side of the body, so-called contralateral hemiplegia. This is the most common motor paralysis of cerebral hemorrhage. A knowledge of cerebral localization will enable one to properly interpret any of the motor symptoms to be found in hemorrhage taking place in other parts of the brain. In most cases of hemorrhage the patient, who may have been standing, suddenly feels giddy, and, after reeling for a few seconds, sinks to the ground or into a chair, and quickly loses consciousness. The physician usually finds his patient in this state, with stertorous breathing, a full, slow pulse, turgid face, and perhaps conjugate deviation of the eyes, that is, with the eyes and face persistently turned to one side.

Diagnosis.—The diagnosis is easy in the majority of cases; at times, however, it may be extremely difficult. One will frequently have to differentiate between this and thrombosis, embolism, and syphilis. In hemorrhage there is a seizure or so-called apoplexy during which the individual is, as it were, suddenly struck down. Convulsions and conjugate deviation of the eyes, loss of consciousness, and Cheyne-Stokes respiration are characteristic of the attack itself. Paralytic phenomena, most commonly hemiplegia, become evident later. The differential diagnosis will be referred to when discussing thrombosis and embolism of the cerebral arteries.

Prognosis.—The prognosis of hemorrhage is grave in all cases. Many patients die in the first attack; many more during a second or third stroke. The outlook is largely governed by the size of the torn vessel and by the situation of the hemorrhage. If the pons or medulla be the seat of the lesion, death is almost certain. If a very large vessel is ruptured, hemorrhage is profuse, and blood may invade either the ventricles or the surface of the brain. In either case the prognosis is hopeless. In most cases of apoplexy with fatal termination death is caused in this manner. Those who have survived the direct effects of cerebral hemorrhage have bled from a small vessel. The outlook in the milder cases is far better than in either thrombosis or embolism. While in the latter softening and cicatrization of the affected brain substance are the cerebral changes, in hemorrhage there is usually only pressure upon the motor tracts. With the shrinking of the blood clot, therefore, restoration of function in the motor tracts may take place even weeks and months after the "stroke."

Extensive meningeal hemorrhages in children and intraventricular hemorrhages in adults are of serious import; death is the usual outcome. Large hemorrhages may become fatal by compression of the brain itself. In addition, it must be recollected that hemorrhages may recur, one of which, if sufficiently extensive, may kill the patient. Besides, the underlying diseases, such as interstitial nephritis and arteriosclerosis, are in themselves serious enough to cause a fatal issue. In many cases "acute bedsore" and intercurrent disease, most often inflammation of the lungs, cause a fatal termination.

Prophylaxis

Prophylaxis will concern itself with the treatment of the underlying causes—arteriosclerosis, chronic alcoholism, syphilis, or kidney disease.

Hygienic Measures.—The patient's mode of life is to be regulated so as to prevent congestion and fluxes of blood to the brain. Anything which may have a tendency to excite the emotions must be avoided. Social gatherings, political activity, and hazardous games of all kinds must be abandoned and exchanged for a quiet, rural, or at least unexciting existence. The diet must be bland; should consist mostly of vegetables, milk, and fruit. Small meals, frequently repeated, are better than few large ones. Spirituous liquors are to be avoided, and physical and mental labor should be reduced to a minimum. A long stay in the country, or in a health resort, is to be recommended. Patients should be warned against the taking of hot, Russian, or Turkish baths. They may bathe in lukewarm water, and can have cool footbaths and so-called sitz baths. Cold douches are not to be used on the head and face. Laxative remedies, such as Carlsbad Sprudel salts, Hunyadi Janos, Friedrichshall water, and other salines, are advised to produce regular daily evacuation of the bowels.

Systematic gymnastic exercise is beneficial in preventing congestion of the brain; provided, however, that all strenuous movements are avoided, especially those requiring the lowering of the head, for reasons that are obvious. Neither heavy lifting nor jumping should be permitted. Those affected with degenerative arterial disease and sufferers from cerebral congestion and increased heart action should not indulge in bicycle riding, ball playing, or rowing. A moderate amount of horseback riding favors the flow of blood to parts other than the brain. The triad, intoxication, constipation, and excitation, is to be shunned by those who have tendencies to cerebral hemorrhage.

Treatment

A patient seized with a stroke of apoplexy, the result of hemorrhage, should be placed in bed with head high and feet low. All tight clothing about the body is to be loosened, and corsets are to be removed. Ice or cloths wrung out in ice water should be placed upon the head and frequently renewed, while the feet are put in vessels filled with hot water. To quiet the heart's action an ice bag may be applied to the cardia. To attract the blood to the intestines a drastic purge may be administered; preferably one or two drops of croton oil in five of sweet oil are placed on the tongue. The patient should be kept perfectly quiet. Movement of any kind is strictly prohibited. Aconite and veratrum viride, in one to two-minim doses every hour, are classical remedies to reduce violent heart action. The suggestions previously given when speaking of cerebral congestion may be here applied with benefit.

In plethoric and robust individuals venesection is indicated. On several occasions I have seen beneficial results from the withdrawal of large quantities of blood in comatose patients. In many cases no good is accomplished by this measure. From ten to twelve ounces (300 to 360 c. cm.) of blood can be withdrawn if the pulse continues tense. The beneficial effects of blood-letting are shown by cessation of convulsions and a return to consciousness. When the hemorrhage into the brain is very extensive, blood-letting will be of no avail. Bleeding seems to do good in the milder cases only.

For the extreme restlessness sedatives and narcotics may be used either by enema or hypodermically. We may give by enema fair-sized doses of chloral hydrate combined with bromids. Morphin may be injected hypodermically in doses of gr. 1/4 to gr. 1/2 (0.015-0.030 gm.).

In those cases in which the diagnosis is doubtful, or rests between hemorrhage and thrombosis, to do nothing is better than to do too much. While waiting for developments several important matters demand the conscientious physician's attention. The bladder must be catheterized once in three or four hours; if this is not done, retention with cystitis is likely to result, which complication is alone sufficient to cause death. The patient's posture requires frequent change in order to prevent hypostatic pneumonia, another complication which frequently carries off the patient, even after he has survived the attack. The lungs require examination within twenty-four hours and frequently afterward. In order to guard against hypostatic congestion and pneumonia, the patient should be turned over to the opposite healthy side, that is, toward the side of the lesion in the brain. This has the effect of facilitating respiration, and tends to prevent the blood from gravitating inward toward the ventricles. A serious danger occasionally following cerebral hemorrhage is "acute bedsore," which must be prevented, if possible, as deaths from this cause are common. By scrupulous cleanliness and frequent change of position, it is often possible to prevent decubitus. When an abrasion is found, aseptic and antiseptic dressings should be applied at once and the patient placed upon an air-cushion or water-bed. No fear need be felt regarding food. If the patient feels hungry, he may be given cold milk for the first three or four days; this will suffice to keep him alive. During the semiconscious state, when swallowing is impossible, peptonized milk may be introduced into the stomach by means of a nasal tube, to which is attached a fountain syringe.

For the rest, symptoms should be watched and combated as they arise. There is sufficient work left for the physician if he attends to bladder, bowels, diet, strict cleanliness, and complications.

A danger that awaits especially the case of apoplexy from hemorrhage during the first few days is the development of cerebritis in the neighborhood of the clot. This complication manifests itself by sudden rise of temperature, convulsions, and a recurrence of the comatose state. The treatment is: antipyretics, ice to the head, cool sponging, and laxatives.

After-treatment.—The after-treatment of cerebral hemorrhage concerns itself with efforts to cause absorption of the extravasated blood and to remove the paralytic phenomena. For the former the administration of small doses of iodids, 5-10 grains (0.3-0.6 gm.) three times daily, has become the classical remedy. For the paralysis massage and electricity are to be employed. The galvanic current applied to the brain was a favorite method in former years, but very few still persist in its use, as galvanism when applied to the brain appears to be entirely devoid of therapeutic benefit, and has done harm in some instances. On the other hand, faradization of the paretic muscles is strongly indicated. Treatment should be begun early, within a fortnight after the stroke, or after the active symptoms of the attack have subsided. Systematic passive and active exercise of the paretic limbs have the highest indorsement. Experimentally it has been proven by H. Munk that passive movements of five minutes' duration twice daily were sufficient to prevent secondary contractures in the paralyzed limbs of animals, while after a discontinuance of passive exercises contractures appeared which did not yield to any amount of passive movement undertaken subsequently. Passive exercises should be begun two or three days after a stroke and be continued for months.

In the application of massage and electricity it is important to remember that only the weak muscles need stimulation; the stronger ones are already too powerful, and, being unopposed, produce the various contractures. Indiscriminate stimulation of muscles is worse than useless. As the extensors of the upper extremity and the anterior and external group of muscles in the lower extremity appear to be the ones mostly affected by the paralysis, these alone should be stimulated. The patient should be encouraged to stand and walk as early as the second or third week after an attack. The sickle-gait in hemiplegia can be largely prevented if patients will make efforts to walk properly, by constantly focussing their attention upon the act of walking, which latter has normally become an automatic function.

The use of splints of cardboard to straighten an overflexed hand and fingers may occasionally overcome obstinate contractures. A splint so shaped as to keep the foot at right angles to the leg is often useful in preventing the contracture of the foot in hyperextension. To be successful all of these measures must be resorted to very early in the case, before deformities have appeared.

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CHAPTER VII

CEREBRAL SOFTENING

(*Encephalomalacia*)

JULIUS GRINKER

1. THROMBOSIS

Etiology.—The underlying conditions which lead to thrombosis of the cerebral vessels are arteriosclerosis and low blood pressure with or without heart disease. The period when both conditions are encountered is old age, as the vessels then become hard and the circulation slow. In middle age syphilis enters as a factor by the production of endarteritic deposits in the arterial coats, favoring clotting within them. The syphilitic variety of hemiplegia is the most common, on account of the extreme frequency of the disease in middle age. Softening of the brain, literally speaking, is more often the cause of death in the aged than is hemorrhage. In some instances, as in the anemias and in some types of septicemia, the cause of thrombosis depends upon changes in the composition of the blood. Blocking of the cerebral arteries may also take place as a result of influenza, scarlet fever, and typhoid fever. In the latter disease we have cardiac weakness with lowered blood pressure, conditions favoring clotting.

Symptoms.—The symptoms depend entirely upon the mode of onset and the locality of the vessel that is thrombosed. When a vessel becomes narrowed gradually, it produces symptoms by diminution of the blood supply to a certain part of the brain, that is, by the production of cerebral anemia. The function of the part is not altogether lost, but merely impaired. Complete restoration of function is still possible when the circulation becomes reëstablished. As a rule, other attacks follow, and eventually complete thrombosis occurs, and with it paralysis of one-half of the body—hemiplegia. The warnings in the form of temporary loss of power in a limb, an eye muscle, or an eyelid are due to the incomplete blocking of vessels. Some of the forerunners of a thrombotic hemiplegia may be a slight loss of memory, transient or partial aphasia; or a patient may complain of tingling, numbness, or weakness in an extremity several days or hours before a complete attack of thrombosis has developed. If a branch of the middle cerebral artery supplying the motor area becomes the seat of thrombosis, there will be loss of power corresponding to the extent of brain affected. In many cases there is gradual extension of thrombosis into other branches of the same artery, and we then have an increase in the paralysis—at first possibly only a monoplegia, later a hemiplegia. In thrombosis loss of consciousness is not the rule, though when a very large artery is the seat of the process this may occur. The usual course is for paresthetic sensations to appear first, then come a slight loss of power, and finally complete paralysis. The convulsions, twitchings, and jactitations which are so frequent in hemorrhage of the brain are uncommon in thrombosis. Symptoms depend entirely upon what portion of the brain is affected; we may have motor or sensory aphasia, hemianopsia, hemianesthesia, hemiplegia, or monoplegia. If the thrombosed artery belongs to the posterior portion of the brain, supplying the cerebellum, we shall have cerebellar ataxia, a reeling from side to side—the so-called drunken gait. We may have nystagmus—a peculiar oscillatory movement of the eyeballs—from interference of function in the corpora quadrigemina, when the vessel supplying them has become thrombosed. Cranial nerve involvement occurs when the affected artery is one that feeds the nerves at the base of the brain.

Pathology.—When a portion of the brain is deprived of its proper blood supply by clotting having taken place in the vessel nourishing it, softening and necrosis are the result. The area so affected may be pale at first, but soon there is an infarct with transudation of blood from surrounding tissues, and the mass may appear red, later yellow. We speak of red and yellow softening according to the coloration produced by the blood. In the later stages a cyst or a scar may be found at the site of a former thrombosis. It must be remembered that, besides softening, which takes place as the direct result of thrombosis, there is edema of the surrounding parts. The symptoms caused by the secondary edema may be more intense than those from direct damage to the brain, but the former soon disappear, while the latter are permanent. This explains the improvement which occurs after a thrombosis, when one would expect none from the irreparable damage done to the brain.

Prognosis.—As the blocking of an artery causes total or partial starvation of that part of the brain which is entirely dependent on it for nutrition, it can easily be understood why the injurious effects of thrombosis are more or less permanent. A mass of brain tissue, once destroyed, can never be regenerated. It is otherwise with the area surrounding the thrombus, in which, as previously stated, there may only be a transient edema. These portions of the brain, when supplied with blood by collateral circulation, may recover function. While the prospects for recovery of function are unfavorable in all forms of thrombosis, this is especially true for the internal capsular area; here the arteries are terminal and collateral circulation is impossible. The result is usually permanent softening. In those cases in which the mind has suffered the prognosis is especially unfavorable.

Physicians have long entertained the view that the prognosis is good in thrombosis due to arterial plugging from syphilitic endarteritis. All that was necessary for their recovery was believed to be the administration of vigorous antispecific treatment. Nothing is more erroneous. A complete aterial blockade—irrespective of how produced—which persists forty-eight hours or more causes death of the part depending upon it for life. How does the prognosis of thrombosis compare with that of hemorrhage? While at first sight an attack of thrombotic apoplexy, with its preservation of consciousness and absence of stormy features, appears less harmful than one of hemorrhage, the facts are otherwise. The prognosis for recovery from paralysis is not as favorable in thrombosis as it often is in hemorrhage. One reason is that the damage to the arteries in thrombosis is more widespread than in hemorrhage. One attack of the former is usually a signal for the recurrence of numerous attacks, while in hemorrhage there may not be any recurrences during a lifetime. For a number of years I have had under observation cases in which only a single attack has taken place; one of these patients had his stroke 20 years ago and is still engaged in a busy vocation.

Differential Diagnosis.—It is always well to remember that thrombosis is the blocking of a vessel with disturbances in the brain centers resulting from lack of blood. Further, that symptoms are produced slowly, for clotting does not take place suddenly, time being required to complete the process. We consequently have a train of symptoms preceding the attack, which may be transient sensory losses, paresthesia, or slight recurring motor palsies. In apoplexy due to hemorrhage, on the other hand, the torn blood vessel permits the heart to pump blood into the brain 72 times per minute; the mass of blood accumulating in the brain causes pressure symptoms almost immediately after the stroke. We only mention convulsions, respiratory disturbances from pressure upon the medulla, Cheyne-Stokes respiration, convulsive twitchings, and, the most important differential symptom, the sudden loss of consciousness.

A case of apoplexy occurring in the young or in an individual under 45 years is mostly thrombosis due to syphilitic arterial plugging. Most *old* people, when struck with apoplexy, suffer from thrombosis. Hemorrhage, on the other hand, occurs with greatest frequency between the ages of 45 and 65. When called to see an apoplectic patient under 45 or over 70 years old, the probable diagnosis of cerebral thrombosis can be safely made.

Thrombosis must also be differentiated from embolism. The embolic attack usually occurs suddenly and resembles in its onset hemorrhage more than thrombosis. Further, the patient affected with embolic apoplexy must have the conditions requisite to produce arterial plugs: an endocardial lesion, aneurysm, or floating clumps of organisms circulating in his blood.

Treatment

While in hemorrhage the treatment aims to retard circulation, lower arterial tension, and to favor coagulation at the bleeding point, in thrombosis we wish to bring about opposite conditions. Here we endeavor to stimulate the heart, raise arterial tension, and accelerate the circulation, so as to lessen the tendency to further coagulation. When the patient is found in a state of syncope, means must be taken to revive the heart's action as speedily as possible. For this purpose nothing is better than hot water bottles applied to the precordial region. Equally important is postural treatment: head low and feet elevated. When consciousness returns, the head and shoulders may be slightly raised, while the bed is lowered. Internally, stimulants must be given, but not too freely, for excessive heart action may cause rupture of a weakened vessel and add hemorrhage to an already existing thrombosis.

Personally I prefer to give one or two tablespoonfuls of brandy internally, and to apply ammonia to the nostrils. This is usually sufficient to revive the patient without overstimulating him.

While in hemorrhage we employ purgatives, salines, and other remedies to deplete the circulation away from the brain, in thrombosis we desire to send as much blood as possible into the brain, and must beware of cathartics. These not only cause the reverse of what is intended, but in addition create an increased coagulability of the blood. After the shock has passed off we may use cardiac stimulants, such as strychnia, digitalis, strophanthus, and brandy.

[Strychnia is not a cardiac stimulant, but it is very valuable in this condition, as it contracts blood vessels, notably those of the splanchnic area, which is followed by increased blood pressure.—Editor.]

In order to prevent constriction in the arterioles of the brain, we add small doses, say 1/100 of a grain, of nitroglycerin to each dose of digitalis or strophanthus. Formerly the arterial dilators, nitroglycerin and sodium nitrite, alone, enjoyed great popularity in the treatment of cerebral thrombosis. We now know that the vasodilators cause a lowering of the systemic blood pressure; consequently they are given almost always in combination with cardiac stimulants.

The treatment of cerebral thrombosis from syphilitic endarteritis is identical with that of syphilis in general. The disappointment which so often follows the faithful application of antispecific remedies to thrombotic cases is due to a non-appreciation of the fact that blocking of a vessel causes irreparable damage to the brain, regardless of the cause that produced it. No amount of antisyphilitic treatment is capable of reviving a portion of brain which has remained bloodless for twenty-four or more hours. Only in the cases in which thrombosis is incomplete, or where a collateral circulation can be established, may recovery take place. Nevertheless, it is our duty in every case of luetic endarteritis causing thrombosis to make use of the most rigid antispecific treatment—preferably by hypodermic injection of gr. ⅛ (0.008 gm.) of corrosive sublimate into the gluteal muscles twice daily, or succinamid of mercury in the same doses. In addition iodid of potassium in large doses, beginning with 30 drops (2 c. c.) of the saturated solution three times daily, and continued by increasing the daily dose 15 drops, distributed over the entire day, until 500 to 600 drops are taken in the twenty-four hours. The last dose is to be continued for about six weeks, then treatment is discontinued for six more weeks. Later, and throughout the patient's life, the mixed antisyphilitic treatment is to be regularly alternated with periods of rest from all medication.

I have stated that coma and loss of consciousness are not frequent in thrombosis. However, when the artery which becomes the seat of thrombosis is large or supplies the medulla, it is possible to have coma and interference with the respiratory centers. This is a condition analogous to that which is observed in hemorrhage, but the treatment differs radically from that of hemorrhage. In thrombosis we must stimulate the circulation so as to send blood into the brain, whereas in hemorrhage we wish to empty the brain of its bloody contents.

In many cases of thrombosis, after the attack has passed off, patients complain of intense headache, which is probably caused by the inflammatory reaction in the vicinity of the softened area. The treatment is: cold affusions to the head, leeches to the temples, and, if necessary, the coal-tar products: antipyrin, phenacetin, aspirin, and salophen, in ordinary doses. If convulsions or delirium appear during the reaction, these measures do not suffice. Thirty-grain doses (2 gm.) of sodium bromid, in combination with 5 grains (0.30 gm.) of chloral hydrate, may be safely administered every four hours until an effect is produced.

The subjects of thrombosis are usually debilitated, worn-out individuals and require nutritious and easily assimilable food. In addition to meat and a liberal diet, small doses of alcohol and stimulating hot broths may be allowed. The stomach, bowels, and bladder all require constant attention on the part of the medical attendant.

The paralyses resulting from thrombosis demand special treatment, and are identical with those from hemorrhage. The after-treatment of hemiplegia is the same in all cases, regardless of the cause which produced it. I shall discuss the treatment of the contractures resulting from the hemiplegic state at the conclusion of the section on infantile cerebral palsy.

2. CEREBRAL EMBOLISM

Etiology.—The most frequent cause of cerebral embolism is acute or chronic endocarditis, principally at the mitral valve. Fibrinous deposits, fresh or old, are there formed, become dislodged, and are swept into the general circulation, reaching the brain. Another factor in the production of cerebral embolism is aneurysm of the ascending arch of the aorta, in which clotting and fibrin formation have taken place. From here fragments may be loosened and swept into the blood current, eventually reaching the terminal or end arteries of the brain. It is also possible for bacterial clumps to block arterioles and thus to cause embolus. Likewise, conglomerations of pigment masses from the destruction of the hemoglobin in malaria may plug a small cerebral vessel and produce the symptom-complex of cerebral embolism. Particles from infected material or fragments of tumor masses, that may have gained entrance into the circulation, may cause either simple or infected cerebral embolism and thrombosis.

The young are more frequently affected than the old, because rheumatism and endocarditis, the two common antecedent factors, are more prevalent in young individuals. In them also the circulation is more active, permitting fragments to be readily swept into the general blood stream. It must be stated, however, that no age is exempt from the development of cerebral embolism.

Symptoms.—From the very nature of the etiology we expect symptoms to begin suddenly. While consciousness is rarely lost—contrary to cerebral hemorrhage—the onset here is abrupt, thus differing from cerebral thrombosis, with its gradual onset and premonitory signs and warnings. In embolism there may be slight twitchings, but rarely convulsions, as in hemorrhage. Neither slight vascular forebodings nor symptoms of cerebral hyperemia and congestion precede embolic plugging. In embolism paralysis develops suddenly, within a few minutes, usually on the right side, and in combination with aphasia. The left side of the brain is commonly selected by the lesion, because it is easier for a plug to reach the brain through the left common carotid—almost a direct continuation of the aorta—than through the right artery, which is a branch of the innominate.

Aside from the difference in onset the permanent symptoms, and even the pathological anatomy of cerebral embolism, are similar to those which have been described in connection with thrombosis. The most common and important symptom is the development of hemiplegia, with or without aphasia, depending upon the localization of the embolus.

Prognosis.—The prospects for recovery are far better in cerebral embolism than in hemorrhage and thrombosis. The patient, being often a young individual with elastic arteries, is not incapable of establishing a collateral circulation. This is not the case in thrombosis, which affects persons with extensive arterial hardening of a kind which does not admit of dilatation for furnishing the anemic brain with nutriment. It must be emphasized, however, that, if recovery in embolism is to occur at all, it must take place soon, for when a portion of brain tissue has been deprived of its blood supply for a few days only, the resulting hemiplegia will be as permanent as in thrombosis and hemorrhage.

Pathology.—The pathological changes resulting from sudden plugging of a cerebral artery by an embolus are almost identical with those occurring in gradual clotting within the blood vessels. There is at first acute softening with subsequent cicatrization, and, in late cases, cystic formation.

Differential Diagnosis.—Embolism is to be differentiated from hemorrhage and thrombosis. We shall take up hemorrhage first. Embolism and hemorrhage both develop suddenly. In embolism, however, there are no premonitory symptoms of cerebral mischief, and the attack is usually not accompanied by convulsions. The patient has suffered from rheumatism and endocarditis of the mitral valve, or is the subject of aortic aneurysm. In any case the diagnosis of embolism is never certain unless the source of emboli can also be ascertained, namely, endocardial disease or aneurysm.

Between embolism and thrombosis there will seldom be difficulties in differentiation, for the latter is usually preceded by symptoms of vascular disease. There has probably been a similar, milder attack, which culminated in a series of slight motor or sensory disturbances. In a young man there may be a history or signs of syphilis. If the attack occurs in a man after 65, with atheromatous degeneration of the arteries, it is probably thrombosis. It is possible for an embolus to become the starting point of a thrombus, and we may then have what is called an embolic thrombosis. In the cases in which there is coexisting heart disease, with low blood pressure and arterial degeneration, the diagnosis between thrombosis and embolism may remain doubtful. The development of a "stroke" during excitement speaks for the diagnosis of embolism, as the latter requires a quickened circulation, while thrombosis is usually accompanied by slow heart action.

Treatment

In embolism it is necessary that the patient be absolutely quiet. An irregular and feebly functionating heart invariably shows a tendency to permit the deposition of fibrin upon the valves, and an overexcited heart washes the fibrin into the general circulation.

As a heart-stimulant I prefer strychnia sulphate in doses of gr. 1/20 (0.003 gm.) every four hours. Occasionally I order tincture of digitalis in doses of 5 drops (0.3 c. c.) every three hours, provided I can watch the patient.

Of course, no hope can be entertained that any amount of treatment will either dissolve or dislodge an embolus. The utmost to be expected is some success in minimizing the amount of thrombosis which often succeeds the embolus. Cardiac tonics are also indicated in all those debilitated states of the heart which allow clots to form within it, as, for instance, in severe cases of typhoid fever and other conditions accompanied by low blood pressure and grave anemia. When the embolus blocking a cerebral artery has originated in a septic source, the prognosis is exceedingly unfavorable. Strenuous treatment will have to be directed not only against the focus in the brain, but also against the original source of infection. From septic emboli solitary or multiple abscesses may form in the brain. The treatment of the end results of cerebral embolus—softening and cicatrization—is the same as in thrombosis.

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CHAPTER VIII

ENCEPHALITIS

(*Acute Non-purulent Form*)

JULIUS GRINKER

Introduction.—The brain tissues, like other organs in the body, are subject to inflammation, which may be acute or chronic, localized or general. Both parenchyma and interstitial tissue may be affected, either alone or in combination. Localized forms of acute encephalitis may occur in connection with meningitis, or follow thrombosis and hemorrhage. Cerebral inflammation in patches may also take place near a tumor, an abscess, or an accumulation of fluid, as in hydrocephalus. Of great importance are the inflammations secondary to acute infectious processes in the vicinity of the cerebral cortex. Perhaps the gravest type of this disease is the variety which occurs in the wake of an acute general infection. In accordance with the best custom, we shall take up the disease under the following subheadings: I. Acute hemorrhagic encephalitis; II. (a) acute hemorrhagic superior polioencephalitis; II. (b) acute hemorrhagic inferior polioencephalitis.

1. ACUTE HEMORRHAGIC ENCEPHALITIS

The disease is mostly always caused by infection. It has been seen in influenza, measles, scarlet fever, pneumonia, whooping cough, and after diphtheria. Inflammation of the brain substance may also be caused by contusion of the brain.

Symptoms.—The disease selects with preference children and young adults. The onset is usually stormy, with headache, vertigo, depression, or irritability. The patient becomes stuporous, semiconscious, and rapidly merges into a coma-like state. Though superficially resembling apoplexy, the coma is rarely profound, and there are no pupillary changes. Instead of a fall there is an immediate rise of temperature, slight at first, but it may become quite high. Paralytic symptoms are not in evidence early, but these usually come on later. In the beginning of an attack stiffness of the neck may appear, rarely general convulsions. There are restlessness, delirium, and stupor. Respiration and pulse are both accelerated; there may be Cheyne-Stokes respiration and slow pulse. In the severe cases symptoms quickly become aggravated and the patient dies in coma twenty-four or seventy-two hours after the onset of the disease. In other instances the course may be protracted, and yet the case terminates fatally even after twenty or more days. If the inflammation is localized in the convexity of the brain over motor areas, we may have epilepsy; if the occipital lobe is affected, hemianopsia may be the result. At the base of the brain encephalitis may cause optic neuritis and other cranial nerve lesions. In those cases in which cerebellar symptoms predominate there are hemiataxia, nystagmus, and rapidly developing optic neuritis. When pons, medulla oblongata, or cerebellum is affected, the symptoms are sufficiently distinctive to direct attention to these localities. Encephalitis affecting exclusively the pontine-bulbar structures is usually described as polioencephalitis inferior.

Pathology.—The acute inflammation has a hemorrhagic character. The affected parts appear hyperemic and swollen, and seem studded with numerous spots resembling flea-bites. Macroscopically, in recent cases, we have the ordinary appearances of inflammation: dilated capillaries and infiltration of leukocytes. In cases that have lasted some time there are found granule cells and extensive proliferation of glia tissue.

Diagnosis.—This is extremely difficult and should, therefore, be made with caution. The symptoms resemble acute serous meningitis, sinus thrombosis, and acute meningitis after infectious diseases. In general it may be said that encephalitis differs from all these affections in the fact that focal signs usually appear early and remain leading symptoms.

Prognosis.—This is extremely grave. When a case develops rapidly with loss of consciousness and high fever, the course is usually a fatal one. On the other hand, certain cases announce quite early that recovery is impending; the stormy symptoms gradually subside and a prolonged convalescence begins, lasting for weeks, months, and even years. Recovery with defect may occur; in fact, this is the rule in the Strümpell type of infantile cerebral palsy.

Treatment

The treatment is purely symptomatic. The patient should be placed in bed and given a quiet environment, as noises and bright lights are harmful. Emotional excitement of any kind should be strictly avoided. Of great value are cold applications or an ice bag to the head; venesection and leeches are to be used early in the sthenic cases. For the fever, if present, we administer antipyretics, but rely principally on cold douching. The bowels should not be neglected. In lingering cases we employ hydrotherapy and electricity. The after-treatment of paralysis is identical with that which results from cerebral hemorrhage, thrombosis, and embolism.

2. ACUTE HEMORRHAGIC POLIOENCEPHALITIS

(a) Acute Hemorrhagic Superior Polioencephalitis.—In 1881 Wernicke first described this type of encephalitis as occurring mostly in whiskey or brandy drinkers, and in some other forms of intoxication. At intervals these patients may have suffered from the symptoms of chronic alcoholism—gastric catarrh, morning vomiting, pains and cramps in the calves of the legs, headaches, amblyopia, weakness and uncertainty in the lower extremities, as well as delirium tremens. Then prodromata appear, such as headache, backache, limbache, vertigo, and vomiting—and perhaps even delirium. Shortly afterward the disease itself appears with acute onset.

The principal symptoms begin either with delirium tremens, or set in suddenly with paralysis. There are states of confusion, restlessness, general trembling and sweating, hallucinations, picking at the bedclothes, or there are the trembling and busy delirium of alcoholics. In addition there may be fever and general weakness. The confusion and motor unrest may have been ascribed entirely to the alcoholism, but throughout the disease delirium is a prominent symptom and a stuporous state becomes more and more noticeable. In some cases the mental condition resembles that of Korsakoff's psychosis. In the cases which are caused by other than alcoholic poisons somnolence appears early. Perhaps the most important symptom of this variety is the appearance either immediately at the onset, or within a few days of it, of complete *ophthalmoplegia.* The ocular paralysis may have been preceded by pupillary inequalities, ptosis, or nystagmoid jerkings of the eyeballs. Another striking symptom, denoting cerebellar trouble, is that the patient's gait becomes peculiar; he walks with feet wide apart and reels from side to side, presenting typical cerebellar ataxia. In most cases muscular weakness is pronounced in the upper and lower extremities. Tremors, like those seen in delirium tremens, and also choreiform movements are often noticed. In addition hemiparesis or hemiplegia may appear; also sphincter paralysis. The tendon reflexes vary; they may be normal, reduced, or exaggerated. In cases with hemiplegia we commonly see clonus and Babinski sign. Dysarthria is a symptom when the pontine and medullary centers are affected. Rarely ophthalmoplegia is seen in association with facial paralysis. The temperature is either normal or subnormal; the pulse is almost always rapid; between 80, 100, and even 140 or more beats to the minute have been counted. The course of the disease is either acute or subacute. Death may occur within a few days or after weeks.

(b) Acute Hemorrhagic Inferior Polioencephalitis.—This is a subdivision of the preceding type, which differs from it in that the gray nuclei of the medulla are mostly or extensively involved. From these nuclei the disease usually extends either upward toward the brain, or downward toward the spinal cord; or there may be only diffuse inflammation of the pons and medulla. The last condition is called acute polioencephalitis inferior or acute bulbar myelitis. While in acute superior encephalitis the ophthalmoplegia is the prominent symptom, in this form—acute inferior polioencephalitis—*bulbar symptoms* predominate. It is the type of polioencephalitis which has recently been observed by Wickman and others in epidemics of poliomyelitis. It was Medin, however, who first saw the true relation between certain isolated cranial nerve lesions and infantile spinal paralysis. Oppenheim promptly acknowledged it, and was one of the first to write on it, so that at present the relation between acute poliomyelitis and polioencephalitis inferior is generally recognized. In the report of the Collective Investigation Committee on the New York epidemic of 1907 we find it mentioned that bulbar forms have been observed among the very acute cases (formes frustes) and in several of the fatal cases. The reporters say: "When the infectious process has been most intense it has been extended into the medulla oblongata and the pons, possibly even to the floor of the third ventricle. . . . Polioencephalitis, then, is an acute infectious process similar to poliomyelitis, of which it may be a part." From the point of view of early diagnosis and correct therapy, it is well to bear this in mind when examining a case of polioencephalitis. By prophylactic measures we may possibly avert an attack of poliomyelitis. While the pontine structures, in which the facial nerve originates, are principally affected, the hypoglossal can also become the seat of inflammation. When pons, medulla, and spinal cord are simultaneously involved, we speak of polioencephalomyelitis.

What follows applies equally to the superior and inferior types of hemorrhagic polioencephalitis.

Prognosis.—There are acute and subacute forms. The acute type generally terminates fatally in from eight to fourteen days, while the subacute variety may pass through a protracted course and not rarely terminates in recovery.

Pathology.—There is a hemorrhagic, inflammatory process, similar in appearance to the one described in the first type of encephalitis. The inflammation is confined principally to the gray matter of the third ventricle and the aqueduct of Sylvius, and may even extend to the fourth ventricle. The process does not limit itself to the gray; white and gray matter may be alike implicated. When the anterior horns of the spinal cord have become part of the disease, as occasionally happens, pathological changes are found in the spinal gray in addition to those of the pons and medulla.

Diagnosis.—For this we consider principally: the onset, which is acute or subacute; the development of focal symptoms, which are mostly phenomena of ophthalmoplegia or bulbar paralysis, or both, combined in various groupings. We also take into account the fact that the disease has a descending course, although the opposite may take place, and that, if the disease be polioencephalomyelitis, it is more or less diffusely distributed over brain and cord. The cranial nerve symptoms yield the clinical syndromes of ophthalmoplegia and glossolabiolaryngopharyngeal paralysis, while the typical spinal disease is a diffuse or circumscribed atrophic cord paralysis. Sometimes cord symptoms predominate over bulbar symptoms; at other times the reverse is the case. The diagnosis should only be made after due deliberation, having regard for all the circumstances attending the development of each case and paying particular attention to the onset, which is that of an acute infection.

Treatment

As the disease has alcoholic or other intoxications and infections for its foundation, we aim first to prevent the further intake of alcohol and of other deleterious substances, and, secondly, to act upon the emunctories so as to cause the excretion of poisonous products from the body. Early in the disease the usual remedies utilized in the treatment of other forms of inflammation are in order. Of these the most important are the various applications of cold to the body, such as ice to the head, general cold water bathing, sponging, etc. As there is a hemorrhagic inflammation present, the remedies advised in the treatment of cerebral hemorrhage also appear to be indicated. It is essential that the patient receive no stimulants of any kind. He should be in bed and avoid all excitement and mental stress. The diet should be nutritious, but non-stimulating. Particular attention must be paid to the possible development of complications. Decubitus may be anticipated by strict cleanliness and the best possible personal hygiene. Bowels and bladder must not be neglected. When hyperemia or congestion are pronounced features, blood should be withdrawn by venesection, and laxatives administered in the manner previously outlined. On general principles, and for the same reasons that they are administered in serous meningitis, iodid of potash and mercurial inunctions have been given. When fever is high the usual antipyretic remedies are cautiously prescribed. For extreme motor excitement, morphin hypodermically, chloral hydrate internally or by enema, and veronal are administered. Hydrotherapeutic measures are helpful to allay the irritability of the nervous system besides reducing the temperature. A good plan is to interchange moderately cold general baths with hot footbaths. Sinapisms to the neck, chest, and extremities may be tried. Lumbar puncture has been made use of therapeutically in numberless cases. The results achieved do not warrant its further therapeutic continuance in this malady.

When the acute disease has become chronic, the remaining paralyses are treated by means of electricity, massage, and orthopedic measures. For the treatment of the poliomyelitic variety the chapter on acute anterior poliomyelitis should be consulted.

3. THE CEREBRAL PALSIES OF CHILDREN

(*Infantile Cerebral Paralysis*)

The paralyses of cerebral origin occurring in childhood may be divided into three groups: (1) paralysis due to conditions arising before birth; (2) those following birth accidents, and (3) palsies dependent upon disease or trauma after birth. They may also be conveniently discussed as (1) the hemiplegic, (2) the diplegic types.

Etiology.—The *prenatal* cases often show deficiency of brain elements—so-called agenesis. Either a portion or all of one hemisphere has been found absent or atrophic. In these cases is seen the peculiar condition of porencephaly, that is, a direct communication between cortex and ventricles, owing to shrinking of the intervening portion, the result of antecedent disease. In other instances there is a lack of physical and mental endurance, capacity for growth is arrested, and the brain succumbs early, having no powers of resistance. In some of these cases the pyramidal tracts have not developed. Even normally the upper motor neurone is formed as late as the ninth intrauterine month, and is not entirely developed until two or three months after birth. In these unfortunates the neurone is probably never finished. Traumatism to the brain of the unborn child very rarely occurs. Hemorrhage or softening has taken place in some cases; in others a meningoencephalitis was the cause. It is admitted by some and denied by others that inherited syphilis lies at the foundation of many cases of cerebral palsy. Illness of the mother during pregnancy has also been made responsible for the causation of cerebral palsy in the offspring.

The majority of cerebral palsies occurring *at birth* are due to difficulties attending the expulsion of the head from the parturient canal. Quite frequently a history of forceps delivery or of protracted labor is the only etiology given. In these cases hemorrhages have undoubtedly occurred in the membranes. The forceps are not always the cause of bleeding; on the contrary, sometimes their timely application has prevented hemorrhage and cerebral palsy. It is hemorrhage in the membranes which causes the largest number of cases of asphyxia of the newborn and stillborn children. From the same cause convulsions and even death may occur soon after birth. If the child survives, the probabilities are that there will be either a hemiplegia or a diplegia later.

Of cerebral palsy following disease or trauma *after birth,* we may mention the vascular lesions, which are also found in adults, such as hemorrhage, thrombosis, and embolism. An additional cause which has of late been prominently brought forward is encephalitis. Strümpell claims that cortical polioencephalitis is the lesion in many of the observed cases. Cerebral venous thrombosis has also given rise to infantile cerebral palsies. It is often a part of a spinal thrombosis, which either precedes or succeeds it. Similar to arterial vascular disease, venous thrombosis may produce softening or sclerotic changes in the motor cortex, with palsy as a result.

Symptoms.—The disabilities following cerebral palsy are not always observed soon after birth, even if the palsy has occurred before or at birth. However, birth palsies are usually noticeable shortly after birth or within a few weeks of it. In the acquired cases the patients appear well up to the development of paralytic phenomena. When first seen some years after birth there is nothing pathognomonic in the appearance of the disease pictures indicative to which category a given case belongs. Possibly the observation that most natal and prenatal cases have been bilateral palsies may aid in diagnosis. The tendency after birth is toward a unilateral paralysis.

The Hemiplegic Type.—Most cases occur in children between the ages of three and six years. The onset is marked by fever, malaise, and convulsions, with more or less disturbance of consciousness. Some time after a convulsion it was noticed, perhaps, that there remained a weakness on one side, more pronounced in the upper extremity. In some cases the motor cranial nerves are also affected. After some time power in the paralyzed extremities may gradually return, but in the great majority of cases much paralysis remains. The preceding symptoms may be classed as early ones. Later symptoms are epileptic fits, mental deterioration, perhaps athetosis, and choreiform movements. When the lesion has occurred in the left hemisphere, aphasia may result. Fortunately children almost always recover speech more or less perfectly, for the right half of the brain appears to act compensatorily. In the hemiplegic cases the arms usually recover less than the legs or face, and the athetoid condition present is mostly confined to the arms.

Cerebral Diplegia.—This form is characterized by a spastic condition accompanied by variable degrees of weakness on *both sides* of the body. In addition to rigidity and weakness there are involuntary movements, but mental deficit here is not the rule. The cases, however, differ widely one from another. In a typical case of cerebral diplegia the movements of the extremities are not as free as they should be. The limbs appear spastic and clumsy. When carefully examined, increased knee-jerks and accentuated Achilles reflexes are found, and, if the child be old enough—over one year—a Babinski sign may also be present. The rigidity in most cases is out of proportion to the weakness. In attempts at walking the attitude is of the well known "scissors-legged" type, owing to spasm of the adductors of the thigh. The arms and face may also be affected. The weakness, always present in some degree, is often masked by the rigidity, which makes voluntary movements more difficult than they would otherwise be. The before-mentioned involuntary movements occur in a considerable number of cases, and often attack the hand and arm, less commonly the leg and face, taking the form known as athetosis, the chief characteristics of which are slow, more or less rhythmical, involuntary movements of the fingers and thumbs, in which hyperextension is a prominent feature. Sometimes either one or both arms participate in movements which may be so violent and uncontrollable as to necessitate strapping the limb to the side. All degrees of mental impairment are met with, from mere backwardness to complete idiocy. Quite frequently there is impaired articulation. Not the least serious symptom is epileptic fits, which are commonly associated with cerebral palsy.

The less severe cases of cerebral diplegia, in which symptoms are confined principally to legs that are rigid and slightly weaker than normal, are usually classed under *Little's disease*, because Little was the first to describe this type. He had in mind cases that are born prematurely and suffer from non-development of the motor tracts, particularly of those for the lower extremities. Such patients may show no mental defects whatever, nor epilepsy; are as a rule undersized, and never learn to walk properly. These patients may have a spastic gait all their lives, but, as their intellect is not often affected, many become useful members of society.

Diagnosis.—The diagnosis is usually not difficult. In those instances in which epilepsy is the prominent symptom, and the evidences of organic disease are insignificant, one may overlook the cerebral palsy and diagnose the cases as essential epilepsy. Careful search, however, will discover slight differences on one side, or there may be bilateral spasticity or clumsiness in locomotion. The admonition cannot, therefore, be urged too strongly that every case of epilepsy in a child should be most carefully searched for evidences of organic cerebral disease.

Prognosis.—The prospects in the cerebral palsies are not good for complete recovery of motor power and intelligence. In the majority of cases some permanent defect is left. The outlook in the congenital cases becomes better the earlier improvement in motion and in intelligence have been noted. In such instances there may be only partial paralysis and little or no mental impairment. In the great bulk of cases, however, there are not only mental retardation, but also idiocy, imbecility, and epilepsy. Most epileptic idiots are of this variety. Cases of Little's disease are among the most hopeful. In these mentality may not be at all impaired and improvement in motion is possible. In the birth palsies the number and intensity of the early convulsions and also their persistence form a rough guide to the prognosis as to life. When slight motion appears in the extremities some months after birth, we may expect still more improvement. There are no criteria to determine what the mental development will eventually be in each individual case. In the cerebral palsies acquired after birth, as a result of disease, the prognosis is much the same as in the cerebral accidents of adults that eventuate in paralysis. One hopeful feature usually not found in the adult is that in cases of aphasia there is, as a rule, return of speech. The prognosis for recovery becomes all the more gloomy when epilepsy persists or is followed by dementia.

Prophylaxis

Under this heading little is to be said. In the interest of the child every pregnant mother should maintain her nutrition to the utmost. The most rigid hygiene should be followed by the mother so as to benefit the unborn infant. In the case of syphilis of the mother a vigorous course of antispecific treatment is indicated in order, if possible, to avert trouble in the offspring. When the infant itself is syphilitic no time should be lost; the little patient must be treated according to the best rules. These cases often do well under treatment, and in many instances the disease has been entirely arrested.

Regarding the birth palsies which are the result of protracted labors, or have been caused by the injudicious application of forceps, physicians are cautioned that it is just as unwise to wait too long for an unaided delivery to occur as it is to apply forceps in every case.

Treatment

Treatment of the Acute Stage.—The principles of treatment of the apoplectic insult, whether from hemorrhage, thrombosis, or embolism, are the same as for adults. As a rule, the pathological cause of the attack in children is not thrombosis, but hemorrhage, and the treatment should be carried out accordingly. Rest, ice-cold applications, leeches, derivative remedies to the intestinal tract, and stimulation of the skin are the most important measures. For repeated convulsions the inhalation of chloroform may become necessary. [Rectal injections of chloral are most valuable, especially in infants.—Editor.] When epileptiform attacks continue to recur, a systematic course of bromids must be instituted. Should the initial stages become protracted and resemble meningitis, treatment will be carried out the same as in meningitis. The after-treatment of an apoplectic insult requires the same degree of care as hemorrhage of the adult.

Paralytic State.—Paralysis is treated on approved lines of therapy. Choreic disturbances are the result of a progressively advancing chronic irritation of the motor tracts. Though we know of no remedy capable of arresting the progress of choreiform movements, nevertheless, we can attempt to remove all injurious influences that may aggravate them. Careful mental hygienic training must be attempted under sensible direction. The child must be enjoined to put forth an effort to inhibit the abnormal movements. For this purpose massage and gymnastics calculated to teach the patient control of his movements are also indicated.

Intellectual defects are often unnoticed until the child begins to develop physically. At a time when children should speak, it may be noticed that they are aphasic or show mental deficit. The aphasia, as has already been stated, is never complete. A certain amount of improvement, even in bad cases, occurs. But much patience is required in teaching the little ones how to acquire speech. After some labor, in the majority of cases, one is rewarded by seeing improvement. Epilepsy is quite common in cerebral palsy; it is a symptom requiring our earnest attention, as the epilepsy itself prevents to a large degree the child's mental development. In many instances such patients remain idiots, imbeciles, or merely backward. Regarding the treatment of epilepsy in cerebral palsy some authorities object to the constant administration of large doses of bromids.

In my opinion, the danger has been overestimated. It is never certain that dementia is caused by treatment. Gowers has long ago taught that the epilepsy itself often has dementia in its wake. While it is true that bromids, continued over long periods of time, may cause acne and even anemia, yet I am convinced that, by judicious administration of ferruginous tonics and the addition of Fowler's solution to each dose of bromid, we can prevent both the acne and the anemia. Of the beneficial effects of bromids upon mentality I have proof in my personal experience. In several instances I decided to give large doses of the bromids to young paralytic idiots. There was nothing to lose and everything to gain. To my surprise I discovered that after a period of several months mental development seemed to progress more rapidly, because the fits had ceased and the patient's general condition improved in every way. This applies not only to Little's disease, when epilepsy is present, but also to acquired hemiplegia. Of course, it is useless to continue the administration of bromids if no benefits result. In those cases, however, in which the attacks are being arrested and no injurious effects are produced upon the gastric functions, it is well to continue the administration of bromids for indefinite periods.

Surgical Treatment.—In cases of infantile cerebral palsy not much is to be expected from operations on the brain. Cases have been operated for epilepsy, chorea, and athetosis. In some of these there has been an improvement in the convulsions immediately after the operation. This improvement has lasted at times six and at other times twelve months.

In my experience the attacks have almost invariably returned. In many cases the operation itself was fatal. Though E. D. Henschen advises operation in cerebral palsy and idiocy, I am not in favor of it, except when undertaken within the first few months after the development of epileptic attacks following cerebral palsy. Even then I do not advise operation, unless focal signs of some kind are present. However, if hemiplegia or monoplegia indicates the side of the lesion and the epileptic manifestations always begin in one extremity, the indication for operation is clear.

For the remote effects of paralysis, namely, late contractures with inability to walk, owing to the spasticity, a new method of treatment has recently been devised by Otfried Förster. The treatment has been tried in a number of cases with remarkable results. Reports from various operators, both here and abroad, are very encouraging, and prompt a continuance of the method. From physiologic reasoning, backed by experiments on animals, Förster concludes that, when the reflex arc in the cord is broken, spasticity—itself an expression of exaggerated reflex activity—must be either reduced or abolished. Further, his past experience has taught him that, whenever to a pyramidal tract lesion there was added posterior cord or root degeneration, existing contractures disappeared. It was by considerations such as these that he was led to devise the operation, which aims to abolish spasticity and contractures by cutting the posterior roots at their exit from the cord. As, according to Sherrington, the sensory root zones overlap from above and below, he advises not to cut two contiguous roots, but to leave intact one or two between each cut root. This he believes to be sufficient to prevent anesthesia. He recommends the operation for the intractable contractures which hinder locomotion, and also for the painful crises from such contractures occurring in Little's. disease, congenital spastic paralysis, compression myelitis, and multiple sclerosis. He insists that only the serious cases should be operated on, and especially those in which spasticity predominates over paralysis. In his opinion it is absolutely necessary to cut at least four roots. In the lower extremities he advises resection of the 2d, 3d, and 5th lumbar roots and of the 2d sacral root. In mild cases only three roots are to be cut. In the upper extremities he recommends cutting the 5th, 6th, and 8th cervical and 1st dorsal roots, or the 4th, 5th, and 8th cervical roots. In cases of so-called contracture crises, in which from time to time the paralyzed lower extremities draw up on the trunk with such violence that the pain is almost unbearable, Förster's operation is the only means of giving relief. His technique is as follows: Free the dura by removal of the arches of the 2d to the 5th lumbar vertebra and of the upper part of the posterior wall of the sacral canal. In order to work with ease the dura should be exposed for at least 2 cm. transversely and be split in the center from below upward. The cauda equina is now completely exposed. As a reliable landmark he gives the fifth lumbar spine, the first sacral root having its foramen of exit on the inner surface of the dural sac opposite this spine. Before removal of any of the vertebral arches, he suggests that a long nail be driven into the fifth lumbar vertebra, exactly one and one-half centimeters laterally from its spine. The nail remains to the end of the operation. When the dura is slightly pulled up at this level one can see the point of exit of the first sacral root. Begin with the second sacral root. Before cutting separate the anterior from the posterior root—the posterior root is recognized by being much larger than the anterior. After having cut the second sacral, proceed to section the fifth lumbar, and, lastly, the third and second lumbar roots. The operation is performed in two stages, on account of the field of operation usually becoming obscured by the accumulation of blood when the dura is opened. Sectioning of the roots can be done several days later. Reports from our busy surgical centers seem to confirm the favorable statistics reaching us from Europe. Regarding the permanency of results, that is, how long the muscles will remain relaxed, the operation is too new to furnish definite data for positive conclusions. It is not to be inferred that the mere cutting of sensory nerve roots is sufficient to restore motion in the parts. The operation constitutes only the first step in a long process of treatment by means of gymnastics, calisthenics, massage, and electricity. The after-treatment is calculated to develop those muscles which have hitherto been unable to functionate because of spasticity and contractures.

Förster's operation requires great skill and should be undertaken only after other measures have failed.

Another advance in the treatment of athetosis and spasticity of muscles incident to infantile cerebral palsy has been inaugurated by Sidney I. Schwab and Nathaniel Allison, both of St. Louis. They published their first article in the *Journal of Nervous and Mental Diseases* for August, 1909, under the caption: "The Surgical Treatment of Athetosis and Spasticities by Muscle Group Isolation." After reviewing the various measures advised for the correction of muscle contractures and spasticity, they come to the conclusion that the isolation of muscles and nerves by means of various strengths of alcohol is preferable to any of the methods in vogue at the present time. In their own words: "We have been led to devise a method which we shall refer to as muscle group isolation. This implies the isolation of muscles or groups of muscles which are at fault in the production of contracture, deformity, or athetosis. It is made effective by cutting off from the central nervous system the connection along which the abnormal impulses, active in causing spasticity or athetosis, are transmitted. This is done by a direct attack upon the nerve itself by isolating it and injecting it with an alcoholic solution. There has resulted in the cases in which it was tried an immediate paralysis of the physiologically stronger groups of muscles, without interfering with the free muscular use of the antagonists. At this point physiological exercises planned to further strengthen the antagonists may be used." They continue: "In the selection of a case on which to try this method for the first time, a simple case of athetosis in which the ulnar nerve is regarded as being primarily involved was chosen, for the reason that the operation would be neither difficult nor dangerous. Inasmuch as this case presented a median nerve complication, it was an easy matter to inject the median nerve at a later time. Our experience in this instance encouraged us to attempt a more complicated operation on a case in which the spasticity was both more general and more intense. Here the condition was bilateral adductor spasticity of the lower extremities in so-called Little's disease, requiring an isolation and an injection of the obturator nerve, which supplies the adductors of the thigh. This nerve descends through the inner fibers of the psoas muscle and emerges from its inner border near the brim of the pelvis. It then runs along the lateral walls of the pelvis above the obturator vessels to the upper part of the obturator foramen, where it enters the thigh and divides into an anterior and posterior branch, separated by some of the fibers of the obturator muscles, and lower down by the adductor brevis. For the purpose of this operation it was necessary to discover the nerve above this division into its branches, that being the necessary point for injection. The fact that this nerve is a motor nerve and supplies a most powerful muscle group, namely, the adductors of the thigh, the gracilis, pectineus, adductor longus, brevis, and magnus, and that this group is all-important in the production of cross-legged progression, made it a most favorable object for testing the value of this operation."

In the *American Journal of Orthopedic Surgery* for August, 1910, Nathaniel Allison, under the heading of "Muscle Group Isolation and Nerve Anastomosis in the Treatment of the Paralyses of the Extremities," again describes the technique for obturator nerve injections with alcohol. In addition, he points out the method of affording relief for overaction and spasticity of the hamstring muscle groups. For this he injects the nerves which supply the biceps, semimembranosus and semitendinosus muscles. These are branches from the trunk of the great sciatic nerve, given off in the upper half of the thigh. Further, he devised an operation for the relief of overaction of the gastrocnemius group. The muscles involved being the gastrocnemius and soleus, which are supplied by branches from the internal popliteal nerve, he injects the latter with alcohol. Lastly, he describes the operation of reaching the anterior tibial nerve, in order to place an injection of alcohol into it for the relief of spasticity affecting the anterior tibial group of muscles. Anyone who intends to perform these so-called "nerve-blocking" operations will find it necessary to read the original article, as the details of procedure are therein fully described.

In a discussion which took place before the Chicago Medical Society, during which Sidney Schwab and Nathaniel Allison had an opportunity to present an account of their alcohol injection method, while Frazier spoke of his results with posterior root section, it was brought out very forcibly that in some instances alcohol injections will be the choice, while in others the posterior root section operation must be selected. Neither of these methods can cause regeneration of nerve structures, or is capable of bringing about restoration of function. The underlying cause being degeneration or non-development of brain tissue, complete recovery is impossible; but great improvement has been noticed both as regards relief from painful contractures, as well as ability to effectively use the extremities. This improvement was the more remarkable, as some of the patients could not take a step before operation, but could get about without mechanical assistance afterward. My own experience with both these methods is limited, but the results I have already attained are sufficiently favorable to warrant further trials.

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CHAPTER IX

CEREBRAL ABSCESS

JULIUS GRINKER

Introduction.—Brain abscess is a surgical affection and belongs especially to otology. The general practitioner, however, is usually the first to see the case in its early stages. Timely recognition enables proper treatment to be instituted and lives to be saved.

Etiology.—Abscess of the brain is either secondary to disease elsewhere, or it is due to infection from without. The microbe at work is commonly the Streptococcus pyogenes or the Staphylococcus pyogenes aureus; the Staphylococcus pyogenes albus has also been found. Other organisms, such as the pneumonia diplococcus, the Bacillus pyocyaneus, and the tubercle bacillus have been frequently observed in the contents of brain abscesses. The infectious source may be situated in a remote part of the body, or it is found in the immediate vicinity of the cranial cavity.

(a) Of the remote infectious sources we mention the following: Purulent depots in bronchi, lungs, pleura; ulcerative endocarditis; peritoneal infections and bone diseases leading to pyemia. The infectious material is often carried by the blood stream and is capable of causing multiple purulent foci in the brain, constituting small or large abscesses.

(b) Of the neighboring sources of infection purulent otitis media takes first place—it being responsible for more than one-half of all cases of brain abscess. Next in order comes suppuration of the scalp occurring after trauma; further, suppuration of the frontal and maxillary sinuses. It is not to be forgotten that carbuncles, pharyngeal ulcerations, or pus formation in the orbital or nasal cavities, as well as purulent meningitis, may serve to transmit microbes to the interior of the brain, causing abscess.

The infectious material may enter (a) through the lymph current by the sheaths of arteries, veins, and nerves, and through the lymph sacs and spaces from the connective tissues; (b) through the blood, by perforating veins, and disease of the bones with or without septic thrombi.

Brain abscesses caused by suppuration in the vicinity of the cerebrum are usually found near the primary infection focus—a fact of great importance in treatment. Such an abscess is usually solitary, or single, and consequently quite operable. The brain abscess proper is often preceded by pachymeningitis externa, with or without extradural abscess. The dura itself is perforated either macroscopically or microscopically and adheres to the pia; it is here that the infectious material enters the brain from without. Abscesses forming in connection with growths occasionally become encapsulated, but later the capsule breaks either in the direction of the ventricles or toward the surface of the brain.

Diagnosis.—The diagnosis of brain abscess is usually not difficult when a distinct source of infection is discoverable. The diagnosis must embrace the following points: (1) Is there an abscess? (2) Where is it situated? (3) Where does it originate?

From the point of view of treatment, the last is the most important question to answer, for it is not sufficient to treat the brain abscess—the primary seat of infection must also be cleared out, or there will be a recurrence. The diagnosis of cerebral abscess must be based upon the finding of, first, that there has been a trauma or other primary source of infection, and, second, that there exist the general signs and symptoms of cerebral abscess.

Symptoms.—The symptoms of cerebral abscess are usually vague and variable. In the *first stage* symptoms of the primary infection still predominate, but there are already signs of extension of the process to the brain itself. This may be indicated by the appearance of *headache, localized pain, vomiting, chills,* and *fever.*

In the *second stage* cerebral symptoms become more distinct. Pain seems to abate, owing to the apathetic condition of the patient; apparently the disease is less threatening, but the patient is becoming stuporous. The temperature is normal or subnormal. Respiration now becomes slow and the pulse is very much retarded, 50 to 60 per minute. Vomiting is not common in this stage, but it may be a symptom at this time. In addition there appears an optic neuritis which is usually not as intense as that of brain tumor. Finally the general signs of infection, such as chills and fever and extreme prostration, are seldom wanting.

In the *last stage,* with the increase in the size of the abscess, the patient grows more stuporous and even becomes comatose.

The localizing signs and symptoms depend entirely upon the situation of the abscess; whether it is developing in a motor, sensory, or a special sense area. Only general symptoms will appear when the abscess is in a so-called "silent" region of the brain. In a general way the localizing signs are similar to those of brain tumor.

From the point of view of treatment we may distinguish the following two principal kinds of cerebral abscess:

1. *Traumatic Abscesses.*—These usually form in the immediate neighborhood of a trauma and are considered purely surgical affections.

2. *Otitic Abscesses.*—They constitute the most important type of brain abscess and belong to otology. Because of their great therapeutic significance they deserve special mention here. In any case of *ear disease* with symptoms pointing to brain abscess all sources of pus accumulation should be freely exposed and drained. If no improvement is noticed after radical surgical treatment of local conditions, a diagnosis of intracranial disease is justified, when either one or more of the following symptoms are present: (1) Headache, often combined with vertigo; (2) vomiting; (3) slow pulse; (4) optic neuritis, from slight engorgement to complete choking; (5) depression of spirits, general apathy, confusion, and somnolence; or symptoms of irritation, such as general or special hyperesthesia, delirium, insomnia, convulsions, or twitchings; (6) paresis of eye or facial muscles.

Occurrence.—Frontal lobe abscess is mostly the result of frontal sinus disease, but may be the consequence of sphenoid trouble; temporal lobe infection is caused by middle ear or antrum disease; cerebellar abscess may be caused by infection from the mastoid cells themselves, either directly or through the intermediary of a sigmoid phlebitis (Macewen).

According to Grunert, 91 per cent. follow chronic and only 9 per cent. acute otitis media. In 9,000 autopsies at Guy's Hospital Pitt found 56 brain abscesses, 18 of which were of otitic origin, while only 1 of them was due to nasal suppuration.

In Macewen's opinion two complications of otitis media offer special difficulties in diagnosis; in one of them symptoms closely resembling intracranial extension of disease may be produced by reflex disturbance through the trigeminus nerve. In the last instance patients show no rise of temperature, but display great tenderness, conforming with the trigeminal sensory skin area. Even the hair cannot be touched without discomfort to the patient. Some of these cases complain of a good deal of headache, nausea, and vomiting, so that the resemblance between this condition and cerebral abscess becomes very striking. However, they recover without operation.

The other complication is serous meningitis. In the latter a lumbar puncture, or a decompression craniotomy, with evacuation of the fluid, proves curative.

Prognosis.—This is absolutely bad without operation; it is not brilliant with operation. In spite of the most improved operative technique, the mortality still remains 50 per cent. This is accounted for by the frequent occurrence of a second abscess due to inefficient drainage. Besides, fungus cerebri, purulent meningitis, septic sinus thrombosis, and pyemia are frequent complications (Macewen).

Prophylaxis

Rhinologists and otologists are alive to the fact that early radical treatment of suppurating processes, while they are extracranial, is still the best treatment of the intracranial complications. According to Macewen, more than one-half of all cases are secondary to suppuration in the otitic and rhinitic cavities. Metastatic abscesses from the lungs may possibly be prevented by attention to suppurating depots in lung and pleura.

Surgical Prophylaxis.—Of the foci near the brain, traumatic injuries occupy an important place. Strict surgical attention must be paid to any trauma applied to the ears, orbit, frontal sinus, pharynx, and antrum of Highmore; these repositories being frequent distributors of the germs causing cerebral abscess.

The possibility for brain abscess to develop, even after a pus depot in the frontal or maxillary sinus has ceased to exist, must be remembered. It is, therefore, advisable to drain freely all suppurating cavities on or about the head, most particularly a diseased ear. Acute suppurating inflammations of the middle ear are rarely the cause of brain abscess. It is mostly the chronic form of middle ear suppuration, with its remissions or intermissions and its tendency to extend to neighboring structures, that has given rise to the worst type of cerebral abscess. No case of chronic suppurating otitis can be considered safe until entirely cured; for, at any moment, and when least expected, a brain inflammation or abscess may flare up so long as there is any pus left in the ear.

When the symptoms indicate that a suppurating process has already extended to the brain cavity, local operative interference is immediately called for. If the signs denote a beginning sinus thrombosis or an extradural abscess with beginning leptomeningitis, radical operation becomes equally urgent. The main object must be, as in skull injuries, to instantly remove infected parts and infectious material, to create free drainage, and to protect the tissues from fresh infection. When minor surgical measures are inefficient to accomplish all this, radical surgery must be employed without delay.

Other prophylactic measures are: timely paracentesis, the removal of polypi and granulation tissue, of carious or suppurating bone, and of necrotic parts. If an extradural abscess has formed between bone and dura, it must be emptied and thoroughly drained. Even the most radical operations may become useless if the process has been allowed to generalize.

Treatment

There is no more treatment by internal medication for a brain abscess than there is for abscess elsewhere. Treatment is entirely surgical. Only when the abscess cannot be localized may one resort to the use of drugs. However, in no case should we rest content with the administration of internal remedies alone, but should be prepared to do surgery at a moment's notice; at least the patient must be under constant surgical observation where immediate operation is possible. It is only where radical treatment for one reason or another is impossible that we are limited to the giving of symptomatic relief. Pain, the most annoying symptom, must be relieved by the usual internal and external remedies.

In the beginning local blood-letting is sometimes useful for the pains of otitis, mastoiditis, and cerebral hyperemia. Leeches or wet cups may be applied over the mastoid processes, the neck, and temporal region. Headache may be treated by an ice cap and cold cloths applied to the shaved head. Vomiting may be relieved by means of chopped ice or small doses of morphin or cocain, hypodermically. The insomnia may be treated by hypnotics, narcotics, or hydrotherapeutic applications.

Operation.—Operation is indicated in all cases of cerebral abscess in which a localizing diagnosis is possible and which can be reached by the surgeon's knife, provided there are no contraindications.

S. E. Henschen gives the following *contraindications* for operation:

1. Abscesses of the multiple or metastatic variety without definite localization. When there is doubt regarding multiplicity, but not concerning the localization which is accessible, an operation may still be performed in view of the otherwise fatal prognosis.
2. When the underlying disease is absolutely fatal.
3. When the patient's general condition does not warrant either an operation or an anesthetic.
4. When the brain abscess has broken through the ventricles.
5. When diffuse leptomeningitis has appeared, or when streptococci appear in the lumbar fluid.

On the other hand, according to the same authority, the following complications do not contraindicate operation: Pachymeningitis, extradural abscess, sinus thrombosis, beginning or localized leptomeningitis, and beginning pyemia.

It cannot be reiterated sufficiently often that while operating for cerebral abscess one must not forget to treat surgically the underlying disease, usually an otitis; otherwise there will be new abscess formation.

Up to the present the results of operation on cerebral abscess have not been uniformly good. As each operator has but limited opportunities and statistics are still meager, the time is not ripe to pass definite judgment upon the value of surgery in cerebral abscess. But even now many successful cases of brain abscess of otitic origin are being recorded. Macewen, when he first published his classic on this subject, had the largest number of intracranial abscesses to report. He had in all 30 cases, of which he operated 24. Of these 23 were cured and 7 died. During the past few years reports on brain abscess operations have become more frequent, especially after Macewen's work had appeared. Körner is correct when he maintains that the cases published so far give an incorrect idea of the value of operation, as only favorable cases seem to find their way into print. In his 1908 statistics, speaking of operative cases in which the abscess was found and evacuated, he mentions 23, of which 11 resulted in cure and 12 died. Of cerebellar abscesses he tabulates 15, of which 4 recovered and 11 died. Of abscesses which were not found he mentions 7 cerebral and 7 cerebellar ones; all of the patients died. After carefully tabulating all the cases in the literature, Körner figures out only 25.66 per cent. of cures. This percentage I think too low for the present time; the diagnosis is now made earlier and operations are not undertaken when a patient is already moribund.

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CHAPTER X

SINUS THROMBOSIS

Julius Grinker

Introduction.—The anatomical peculiarities of the venous sinuses favor clotting within them. Their lumen is irregular and triangular in shape; the walls contain no muscular tissue, and, being imbedded in the substance of the dura mater, their caliber does not vary. When the circulation becomes feeble, as, for instance, in cases of prolonged diarrhea, the blood flow through the sinuses becomes retarded, and thrombosis can readily take place. The superior longitudinal sinus is peculiarly prone to thrombosis, because the veins emptying into it ascend from the surface of the brain, entering in a forward direction, which is directly opposite to the flow of the sinus itself. There are two types of sinus thrombosis. One variety results from conditions of malnutrition and prostration, and is called marantic or primary sinus thrombosis. The other—the more important—is due entirely to extension of infections into the sinuses from other regions; it is, therefore, described as secondary or infective thrombosis.

1. MARANTIC OR PRIMARY SINUS THROMBOSIS

The superior longitudinal sinus is the seat of predilection for this variety. It is more common in early childhood than in adults, excepting those who are senile and feeble, in whom it not infrequently occurs. In infants prolonged attacks of gastroenteritis and bronchial disease predispose to this condition. Extreme states of exhaustion following infectious diseases, such as measles, scarlatina, typhoid fever, and diphtheria, can give rise to this variety of sinus thrombosis.

Symptoms.—In a general way the symptoms resemble those of cerebral hemorrhage. There are in both irritation and paralysis phenomena. The disease has similarities with meningitis, fever being present in both; also optic neuritis and contracted pupils, nausea, and vomiting.

In thrombosis of the transverse sinus edema appears in the soft parts of the mastoid region. The external jugular vein is unequally distended on the two sides. When the thrombus is continued into the internal jugular vein the latter feels as a tight cord, while the soft parts of the neck are swollen. The head is inclined to one side and its movements are accompanied with pain. There are no distinct cerebral symptoms in this type of thrombosis, owing perhaps to the circumstance that blood still flows in the opposite transverse sinus. Thrombosis of the cavernous sinus manifests itself by symptoms of stasis in the orbit, such as swelling of the lids and face, as well as edema of the optic nerve, and sometimes even by paralysis of the ocular muscles.

Prognosis.—The prognosis is quite serious in the majority of cases.

Prophylaxis

The etiologic factors capable of producing the disease should receive careful attention. First of all, heart weakness—the immediate cause of thrombosis in the sinuses—demands thorough treatment. In the presence of brain symptoms and in hydrocephaloid conditions generally we must beware of the depressing narcotics and the withdrawal of blood from the vicinity of the head. These measures, undertaken, perhaps, for other conditions, prove dangerous by favoring thrombosis. In addition, if it is desired to prevent thrombosis, the heart must be reinforced.

Treatment

General Treatment.—Stimulants should be administered with a free hand—wine, ether, camphor, spirits of ammonia, and ether injections. Sinapisms over the heart and the peripheral parts are in order. Hot baths are not permitted, because of the cerebral anemia produced by them.

Local Treatment.—One serious danger to be avoided is cerebral hemorrhage, which frequently follows sinus thrombosis. For this, local bleedings are indicated to reduce blood stasis. Nature sometimes shows us the way by causing epistaxis. Leeches may be applied to nose, forehead, temples, and the mastoid region, and wet cups over the neck. Generally speaking, the treatment is that of cerebral hemorrhage. When convulsions and delirium are present narcotics are indicated. After the attack has passed off, and when paralysis, somnolence, and coma appear, stimulating remedies are applied both internally and externally. Paralyses are treated in the same manner as those resulting from hemorrhage, thrombosis, and embolism.

2. SECONDARY OR INFECTIOUS THROMBOSIS

Etiology.—Frequently the infection is mixed. Several kinds of germs are met with: streptococci, colon bacilli, and pneumococci. The primary cause is usually a chronic purulent otitis media. Rarely is an acute case with pus retention the cause of the infection. Infectious sinus thrombosis may be secondary to thrombosis elsewhere. In the majority of cases thrombosis is an indirect result of mastoid suppuration. The anterior wall of the sinus is first attacked; somewhat later the sinus itself becomes affected. The right is more often implicated than the left side, for the reason that the sigmoid fossa is larger and extends more anteriorly and outwardly than the left side; the wall of the inner ear being also thinner on the right side. Additional causes are: injury to the cranial bones, osteomyelitis, tuberculous or syphilitic caries, and suppuration of the scalp.

Pathology.—The affected sinus is distended and feels as hard as a cord. A clot adhering to the walls of the sinus usually fills its lumen. In this type of sinus thrombosis the clot may quickly break down into pus, and general pyemia may be a consequence. The ventricular fluid is usually increased, and extensive softening of the brain may occur if this be long continued.

Symptoms.—The symptoms may be divided into three groups: (a) cerebral symptoms indicative of some intracranial disturbance; (b) local signs revealed by external examination of the head; (c) general signs of some bodily condition with which sinus thrombosis is likely to be associated.

Cerebral Signs.—The brain symptoms of infectious sinus thrombosis differ in no way from those of other intracranial lesions. There may suddenly appear attacks of vomiting, convulsions, and coma, which rapidly terminate in death. In other cases the symptoms are not as stormy; there are headache and restlessness, followed by delirium, and sometimes later convulsions and coma. Occasionally there are present the symptoms of meningeal irritation—rigidity of the neck muscles, trismus, unequal pupils, strabismus, nystagmus, irregular pulse, and respiratory disturbances.

The *local signs* of sinus thrombosis differ with the situation and the degree of clotting in each case. Their presence only is of value; their absence cannot be considered as negative signs for diagnostic purposes. In disease of the superior longitudinal sinus there may be an edema of the forehead to attract attention to this place. In rare cases the temporal veins may be distended and even thrombosed. Relative emptiness of one side of the superficial veins as compared with those of the other side of the head, which may be distended, favors the diagnosis of lateral sinus thrombosis in the latter. The jugular vein may be felt as a solid cord, very tender on pressure. When the cavernous sinus is affected there is usually slight proptosis of that side, with edema of the conjunctiva and of the upper part of the face. Amblyopia is the rule; the ophthalmoscopic picture shows swelling of the disc, or perhaps thrombosis of the central vein, with multiple retinal hemorrhages. Thrombosis may extend from sinus to sinus; in fact, it is quite the exception to find post mortem that the thrombus has limited itself to one sinus.

Prognosis.—In the majority of cases the disease, if left alone, has a tendency to become rapidly fatal. Cases of infected thrombosis following ear disease have been saved by early surgical interference—tying of the jugular vein and the affected sinus on either side of the thrombosed area—the sinus being later incised and the clot removed. Where pyemia is present, either as a cause or the result of infected thrombosis, the termination is always fatal.

Prophylaxis

The prevention of infectious sinus thrombosis is almost identical with the prophylaxis of brain abscess. Every infection near the cranium and elsewhere in the body must be carefully investigated and treated. Even an insignificant trauma to the head is to be considered as fraught with serious possibilities, unless treated according to the most approved rules of asepsis and antisepsis. Likewise, inflammations about the ear, nose, and throat are not to be lightly considered, especially if the inflammation be a purulent one or is likely to become one. While all kinds of inflammations in the nose and its accessory cavities are of great etiological importance as regards the development of infectious sinus thrombosis, nothing surpasses in gravity chronic purulent otitis media. It is necessary, therefore, to pay strict attention to this source, not only after symptoms of brain abscess have appeared, but long before there is any sign of cranial mischief. At no time during the continuance of a purulent chronic otitis media should surgical intervention be declined. All the more is this true when the first signs of extension to the mastoid cells have made their appearance. Then everything possible should be done to rid the patient of the pus depots which threaten his life.

Surgical Treatment

As nothing can be expected from internal medication, it becomes imperative to resort to radical surgery as soon as possible. The object is to lay open the infectious sinus depot, clear out and drain it thoroughly, and at the same time to evacuate any primary focus which may be found. If the latter has not yet been discovered, every effort should be made to find it. Of the various sinuses we shall speak in particular only of the lateral sinus, because of its great importance.

Thrombosis of the *lateral sinus* is, unless treated, a fatal disease. It is necessary to open the infected sinus in connection with the mastoid and to remove all infectious débris. The diseased tissue between the primary infectious source and the sinus must be removed *in toto.* Especial attention is given to the extradural collections of pus and suppuration in the sinus wall, which are often the mediators or the direct cause of sinus thrombosis; their timely evacuation often prevents the formation of an infected thrombus. Thorough work in this direction is capable of preventing the further growth of an already infected sinus.

As it is usually impossible to prevent the spread of infection through the jugular vein, it will be safe, according to Horsley, to tie the vein before opening the sinus. This is also necessary for the prevention of hemorrhage. According to Jansen, however, tying of the jugular vein is only indicated when the thrombus extends into it. According to the statements of operators, not much is to be feared from pyemia originating in the proximal end of a thrombus.

Contraindications for operation are: advanced tuberculosis, marasmus, grave sepsis, and especially those diseases which are fatal in themselves.

Results of Operation.—Not a sufficient number of cases have been published to definitely determine what a radical operation will accomplish for infectious sinus thrombosis. Besides, there is a tendency for operators to publish their favorable cases, leaving out those which are apparently of no interest, namely, the fatal ones. From our point of view, this is a mistake.

In comparing the earlier statistics as furnished us by various authors from different clinics, we learn that the majority of surgeons have had on an average 50 to 60 per cent. of recoveries, with the exception of Macewen, who can show 72 per cent. of cures. Other operators speak of 61 per cent. cures. One author, Chipault, cannot boast of more than 50 per cent. recoveries. In Körner's last statistics we read of 74 to 77 per cent. of cures.

It goes without saying that results depend greatly upon the state in which the patient is found prior to operation, and also upon what complications were present. If an operation can be done before pyemia has occurred, recovery may be expected, unless there are other fatal complications present, such as leptomeningitis, brain abscess, etc. It is always advisable to operate as early as possible before signs of septicemia or pyemia have developed. It would also appear that the tying of the jugular vein seems to exert a favorable effect upon the prognosis, for in the cases in which the vein had not been tied the rate of mortality was higher than in those in which this was done. The cause of death in most cases was either pyemia, brain abscess, or hemorrhage.

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CHAPTER XI

TUMORS OF THE BRAIN

Julius Grinker

Introduction.—Ever since surgery has invaded the cranial cavity the subject of brain tumor has assumed immense practical importance. It becomes necessary, therefore, to give adequate space to the diagnostic criteria upon which a rational therapy must be based. For these we are indebted principally to the labors of Bruns, Oppenheim, and Henschen, while in the surgical field most elucidating contributions were made by Horsley, Krause, Cushing, and a host of others. In the preparation of this chapter the writings of all these authorities were freely consulted.

Definition.—A brain tumor may be defined as a growth in the cranial cavity originating from the brain, membranes, cerebral blood vessels, or cranial bones. Ordinarily by brain tumor is meant a solid growth, though cysts of various kinds are usually included in this definition.

Before entering upon a discussion of treatment, it is necessary to briefly review not only symptoms and diagnosis, but also the pathological anatomy.

Varieties.—The following anatomical kinds of brain tumor will be considered: (1) tuberculoma; (2) syphiloma; (3) endothelioma; (4) glioma; (5) sarcoma; (6) cysts; (7) carcinoma; (8) benign tumors of different kinds.

(1) *Tuberculomata* are growths consisting of conglomerations of tubercles of varying size, which usually appear as single or solitary tumors. This type of neoplasm occurs most often in the cerebellum and, according to Allen Starr, is most frequent in childhood. Only those varieties of tuberculoma having a fibrous envelope are capable of radical removal.

(2) *Syphilomata* represent the type of infectious granuloma oftenest seen in the adult. Of all syphilitic new formations these are most resistant to antiluetic treatment; they may reach a large size, and are often multiple. In many cases, surrounded by a dense fibrous sheath, and superficially placed in the brain, syphilomata have been successfully treated by the surgeon after medicines had failed to produce any marked change.

(3) *Endotheliomata,* also classified as fibrosarcomata, and formerly

considered as true sarcomata, constitute the most frequent form of nonspecific benign cerebral neoplasm. These tumors originate in the meninges, are encapsulated, and do not form metastases. As their effects upon the nervous substance are exerted by pressure rather than infiltration, brain functions may be restored to normal after their complete removal. Readily accessible in most cases and easily shelled out of their capsules, these tumors are best treated surgically. Their favorite localization is the cerebellopontine recess on either side.

(4) *Gliomata.*—Tumors originating from the neurogliar tissues appear in two distinct forms—the hard and soft gliomata. The latter, often possessing but little more consistency than brain tissue, have a tendency to infiltrate the brain mass to such an extent that their borders cannot even be microscopically determined. They may reach an enormous size and are often transformed, in whole or in part, into cysts. Further, these tumors are exceedingly vascular and hemorrhages occur in them, which can be mistaken for ordinary apoplectic attacks. The other variety —hard gliomata—are occasionally surrounded by a false capsule, making possible their successful separation from the remaining tissue and their consequent removal.

(5) *Sarcomata.*—These tumors are generally of firmer consistency than most gliomata, and can be easily distinguished from the surrounding tissues. When encapsulated they can be shelled out of their covering. The tendency for sarcoma is to spread to adjacent tissues and to multiply by metastasis. Sarcomata are subject to regressive metamorphoses; they either become cystic or undergo mucoid degeneration, forming so-called myxosarcomata. Like sarcoma in other parts of the body, they are not always removable and show a tendency to recurrence. Generally speaking, the symptoms are those of compression, unlike the gliomata, which cause symptoms by infiltration.

(6) *Cystic growths* are either the result of parasitic activity, as from cysticercus and echinococcus, or else they follow trauma. They may occur in any part of the brain, and have also been observed in the fourth ventricle. As previously stated, gliomata not uncommonly degenerate into cysts.

(7) *Carcinomata* are always of metastatic origin. Usually the metastases lodge first in the cranial bones, and later invade the cranial cavity itself. This is equally true of sarcoma.

(8) *Benign tumors* of different kinds occur, but are not common. Examples of each of the following have been reported in the literature: fibroma, myxoma, psammoma, osteoma, cholesteatoma, lipoma, and teratoma.

Etiology.—The true causation of brain tumor is as little known as that of tumor elsewhere. It is certain, however, that, of all predisposing and exciting factors, trauma occupies the first place. Following an injury to the cranium there may be a latent period during which no symptoms are present, and the incident may have been forgotten; after a variable period of time, however, a neoplasm is discovered at the former site of trauma. In this way originate the so-called osteoma and osteophytes. Not only bony tumors, but also those springing from the membranes, have trauma as their exciting cause. The histological makeup of neoplasm following injury often depends upon a patient's constitutional tendencies; in a syphilitic, gummata are likely to appear, while in the tuberculous, the so-called solitary tubercle is more apt to develop.

Situation.—Though a tumor may be situated anywhere in the brain, yet certain types are more constantly found in definite locations. Thus, tuberculoma is common in the cerebellum, syphiloma in the basal meninges, endothelioma in the meninges of the subtentorial region, while cysts are frequent in the cortex, cholesteatoma in the temporal lobes, teratoma in the pituitary body and at the base of the brain. It must be remembered that tumors are not always found in the situation in which they originated, as displacement not rarely occurs.

Brain tumors are divided into *benign* and *malignant* forms. Among the *benign* varieties, also called primary tumors, are named: endothelioma, fibroma, lipoma, osteoma, cholesteatoma, psammoma, myxoma, angioma. As *malignant* tumors are mentioned the metastatic growths, carcinoma and sarcoma. The division is important from the therapeutic point of view, the metastatic growths for obvious reasons being inoperable, while the primary, or benign, tumors, appearing as solitary growths, are proper objects for surgical intervention.

Symptoms.—Not all brain tumors produce symptoms, and it is not rare for a latent tumor to be discovered on the operating or post mortem table. This may be accounted for by the tumor either being too small to produce symptoms, or else, growing very slowly, the surrounding tissue becomes gradually accustomed to the new growth. Another reason might be the situation of the tumor in a "silent" area, such as the frontal, right parietal, or right temporosphenoidal lobe, all of which are still silent in respect to their functions.

The manifestations of brain tumor are divided into (1) general symptoms, due to progressive increase of intracranial tension common to the majority of tumors, and (2) special or focal symptoms, depending upon the portion of brain involved. The last often enable an exact localizing diagnosis to be made.

General Symptoms.—The general symptoms of brain tumor are still the classical three: (1) headache, (2) nausea and vomiting, and (3) choked disc; but several others may now be added to this triad, namely, (4) vertigo, (5) slow pulse, and (6) convulsions. These are the important ones. They may be present while localizing signs are still absent. This is particularly the case when a tumor, situated in a "silent area" of the brain, affects cerebral functions too obscure for our detection. It is also possible for a small tumor to so obstruct the aqueduct of Sylvius that there ensues internal hydrocephalus, yielding general symptoms, but no localizing ones. Likewise, by reason of the pressure exerted upon the entire brain mass, subtentorial tumors early produce general symptoms, which may be long delayed in frontal lobe tumors.

Headache.—Cephalalgia is an early symptom in most cases and is especially intense in tumors of the cerebellum accompanied by internal hydrocephalus. Severe localized headache is also frequent in superficial cortical tumors. Headache is mild when a neoplasm is small or localized in the white substance. On the other hand, there may be intense neuralgic pains when a growth presses upon such a sensory structure as the Gasserian ganglion.

Nausea and Vomiting.—Rarely is there an entire absence of these symptoms. Most frequently seen in tumors of the posterior fossa when the vagus region is directly compressed, they are also observed in neoplasm situated in other parts of the cranial cavity.

Choked Disc.—This is a symptom characteristic for brain tumor, though also seen in abscess, hydrocephalus, etc. According to Oppenheim, optic neuritis is found in 90 out of 100 cases. It is usually bilateral, rarely unilateral; often one side is more affected than the other. Choked disc not being present in all brain tumors, its absence is less valuable than its presence. In the pons it is so frequently absent that this negative finding has almost attained to the dignity of a localizing sign. In the early stages optic neuritis may not be accompanied by visual disturbances. When a neuritis merges into atrophy, vision invariably suffers. Choked disc is an early and rapidly developing sign in cerebellar tumor, which is often followed by sudden blindness. With Sänger and Cushing, I am of the opinion that the process is due to an edema and should be called papilledema.

Vertigo is a symptom depending for its production mostly upon disturbance of the vestibular mechanism.

Slow Pulse.—This is usually a late symptom, and may be accounted for by pressure on the vagus nucleus, causing irritation of the cardiac center.

Convulsions.—Convulsions are classed among the general signs of brain tumor, because they occur with cerebral neoplasm regardless of location. The mechanism is explained by pressure upon cortical cells causing constant irritation with subsequent motor explosions.

Special or Focal Symptoms.—Varying with each location, these symptoms enable us to determine the exact seat of a tumor. To explain in detail all of the focal signs would be equivalent to discussing the entire anatomy and physiology of the brain. Only important points will, therefore, be touched upon.

Focal signs may indicate a tumor in the psychic, sensory, motor, or vasomotor brain territories. We make a distinction between irritative and destructive symptoms. An *irritative* symptom is one which causes hyperfunction; for instance, when present in the sensory sphere, there will be pain; in the motor region, twitchings or convulsive movements. A *destructive* symptom, on the other hand, is one which paralyzes the part; instead of hyperfunction there is absence of function, and the result is motor, sensory, or psychic paralysis. From the point of view of diagnosis, irritative symptoms are of the greatest importance. They enable us to infer that a growth is beginning to impinge upon a certain portion of the brain. Because paralytic symptoms are usually late in appearing and may be caused by neoplasm in distant parts of the brain, they have no localizing value by themselves, but, occurring in conjunction with irritative symptoms, are almost pathognomonic.

Diagnosis.—A correct localizing diagnosis can only be made by careful systematic examination, which includes both negative and positive findings. For a correct pathological interpretation of brain tumor symptoms, one must inquire diligently into the condition of lungs, stomach, kidneys, prostate, and testicles. This conforms to the rule in neurology that, in order to make a pathological diagnosis, organs other than those of the nervous system must be carefully interrogated.

Differential Diagnosis.—A consideration of the etiology, the progressive afebrile course, the presence of choked disc, intense headache, as well as the symptoms of gradually increasing brain pressure, should protect one against mistaking a brain tumor for any other condition.

It must be recalled that not every case of optic neuritis with intense headache means brain tumor, for these symptoms may also occur in chlorosis, polyneuritis, and chronic nephritis.

General paresis under certain aspects resembles brain tumor. The absence of papilledema, headache, and vomiting, and the presence of a positive Wassermann reaction, will decide the diagnosis in favor of paresis.

Hydrocephalus is with great difficulty differentiated from certain types of brain tumor. In some cases the diagnosis is impossible. This will be better understood when we recall that the following symptoms are common to both: headache, optic neuritis, nausea, vomiting, vertigo, stupor, and a chronic progressive course. Tumor may be differentiated from hydrocephalus by the possible presence of focal signs and the history of the case. Hydrocephalus is often preceded by an acute infectious disease, as influenza, scarlet fever, etc. In addition, large size of skull, remissions followed by fever, are symptoms indicative of hydrocephalus. Lumbar puncture will help to determine whether the cerebrospinal fluid is under increased pressure—a symptom of hydrocephalus. A note of warning must here be sounded against the indiscriminate use of lumbar puncture. Several deaths from this procedure have already been recorded in tumors of the posterior fossa, especially of the cerebellum. And it is precisely this variety of tumor which must occasionally be differentiated from hydrocephalus. In such cases it is best to rely upon other differential points.

Pachymeningitis interna hæmorrhagica has many symptoms resembling brain tumor. The etiology of alcoholism or psychosis in the former, as well as focal signs not of the convexity, may also help in differentiation. The fact, however, remains that hematoma is practically a tumor and is treated as such.

Idiopathic *epilepsy* may likewise cause diagnostic difficulties. The absence of optic neuritis and of focal signs favors epilepsy as against tumor. Brain tumor with Jacksonian fits may be mistaken for Jacksonian epilepsy from other causes. This is particularly the case when the growth is cortical and there is no papilledema present. Here the other general symptoms must aid in the differentiation.

Brain abscess has many things in common with cerebral tumor, but it differs in etiology and symptoms develop more rapidly.

Having made a diagnosis of brain tumor, the next step is to *localize* the same.

Regional Diagnosis.—In attempting a localizing diagnosis of the seat of a brain tumor particular attention is paid to the earliest symptoms of irritation and destruction. Careful inquiry should be made relative to the first appearance of localized spasm and paresthesia; for tumors may not cause any other symptoms for a long time. Not only positive symptoms, but also negative findings, are utilized in making the localized diagnosis. Only by excluding every other condition in a given case does the diagnosis become firmly established.

Central Convolutions, Right Hemisphere.—Clinicians are now pretty well agreed that the ascending frontal (precentral) convolution contains the motor area, while the ascending parietal (postcentral) convolution in either hemisphere is the seat of the sensory centers. As a rule, tumors localized in the central convolutions cause both irritative and destructive symptoms.

Motor irritative phenomena appear in the form of localized spasms or tonic muscular contractions, which affect more or less constantly certain muscle groups or movements, such as flexion and extension. The tendency for localized twitchings is to successively spread to the nearest group of muscles. In the beginning there may be twitchings of the fingers only; later the movements extend to the muscles of the forearm, arm, shoulder, neck, and face; still later to the trunk and leg muscles, but always following the anatomical arrangement in the motor convolutions. The entire half of the body may in this way become violently convulsed. Subsequently the other side may become implicated in the reverse order.

The sensorium is usually clear in the lighter grades of Jacksonian epilepsy, but in severe attacks there may be partial loss of consciousness. Of great value for diagnosis is an accurate account of how the convulsions begin. Such knowledge practically amounts to the making of an exact localizing diagnosis. According to the late Hughlings Jackson, who first described them and whose name they bear, the localized convulsions occur only in lesions of the central convolutions. Clinical experience and animal experimentation have fully confirmed the truth of his assertion.

Motor paralysis may succeed a Jacksonian fit, or it may appear in parts that have never been the seat of spasm. One arm or one leg usually becomes paralyzed first; rarely does the paralysis begin in the face. Frequently the disability in the arm is more pronounced than in the leg.

As previously stated, isolated paralysis may be produced by pressure from a distance, in which event it has no localizing value. On the other hand, muscular twitchings recurring in the same parts and followed by paralysis in these parts, constitute an important focalizing sign.

Sensory irritative phenomena appear in the form of localized paresthesia, such as formication, pain, or disturbances of temperature. They have an importance for local diagnosis similar to that of the corresponding motor phenomena. The more localized the paresthesia the greater is its value as a localizing sign. Sensory disturbances often precede the motor symptoms by a considerable length of time.

Sensory paralysis—anesthesia—is usually not limited or circumscribed, as, for instance, in the corresponding motor disturbance. The more rapidly an entire brain center becomes affected the more pronounced is sensory paralysis. On the other hand, in a slowly growing tumor anesthesia is usually ill defined.

When both motor and sensory irritative phenomena are present, one is justified in localizing a tumor in the central convolutions. On the other hand, the absence of irritative and paralyzing symptoms in either the motor or the sensory sphere almost excludes the existence of tumor in these convolutions and their immediate vicinity. It is to be remembered, however, that a slow-growing tumor in one motor area may not cause any motor symptoms, perhaps because the other hemisphere compensates. The question in any case of motor or sensory hemiplegia often arises: "Is there neoplasm growing within the central convolution sphere or elsewhere?" The history of the case may offer valuable help. Tumor starting in the precentral convolution or its vicinity may cause monoplegia first, and only later hemiplegia; while, in tumors lower down, as of the internal capsule, hemiplegia is more complete and usually develops more rapidly.

Frontal Lobe.—The localizing symptoms of tumor in this situation are: (1) constant frontal headache which is accurately localized; (2) tenderness upon percussing the frontal bone—valuable only when present. In some cases there are (3) mental signs closely resembling general paresis, such as apathy and complete mental inertia, weak memory, lack of initiative, depressed or exalted mood. In tumors situated on the under surface of the frontal lobe we have: (4) symptoms of anosmia on the same side as the tumor; (5) symptoms of pressure upon the chiasm, optic tract, or optic nerve, causing bitemporal or homonymous hemianopsia or unilateral amblyopia. An interesting symptom in connection with frontal lobe tumor is (6) frontal ataxia, a reeling from side to side, which differs in no essential from the well-known cerebellar ataxia.

The Parietal Lobe.—When the superior lobule is implicated, the stereognostic sense may be disturbed, that is, the patient is unable to recognize objects by mere touch. When the tumor encroaches upon neighboring parts, symptoms will appear calling attention to their respective areas: from the postcentral convolution, sensory paralysis; from the first temporal convolution, auditory hallucinations; from the occipital lobe there will be visual disturbances of a kind similar to those from occipital lobe diseases. Other symptoms are: localized headache and tenderness on pressure over the parietal lobe. The right parietal lobe, being regarded as a silent area, is recommended by Sänger as the place of choice for decompression operations.

Temporal Lobe.—Though the diagnostic signs of tumor in this lobe are neither marked nor reliable, yet the following points deserve mention:

Contrary to what one may have expected, there is no defect or loss of hearing when the auditory center on one side is destroyed, audition having a bilateral innervation in the brain.

When the lesion is in the hippocampal gyrus and near the tip of the temporal lobe, there are slight olfactory and taste disturbances. However, tumors implicating the cortex of either the uncinate or the hippocampal gyrus are known to produce certain seizures, the so-called uncinate group of fits (Hughlings Jackson). Typical examples of this condition have been studied by Purves Stewart, Mills and Cushing, who have seen six cases each. The uncinate seizures are characterized by peculiar sensations of smell and taste, usually of a disagreeable quality. There may also be an epigastric aura. With the sensory disturbances there may be motor phenomena, such as chewing and swallowing movements, also salivation; consciousness usually being retained. The attacks may be replaced by, or be associated with, so-called "dreamy states," viz., vague feelings of the unreality of surrounding objects.

When a tumor is situated in the posterior portion of the temporal lobe, near the first temporal fissure, or the second temporal convolution, there may be diminution of vision or homonymous hemianopsia without hemianopic pupillary reaction—a neighborhood symptom from the occipital lobe. Similarly, quadrantic hemianopsia has been observed in tumors of this region.

Neoplasms of the anterior median division of the temporal lobe near the hippocampus may, by pressure upon the posterior portion of the visual tract, produce homonymous hemianopsia with hemianopic pupillary reaction, another neighborhood symptom.

LEFT HEMISPHERE.—By reason of the presence of speech centers, localizing diagnosis in the left hemisphere is comparatively easy. Though there are many varieties and a number of types of aphasia, for our purpose it is only necessary to review the three classical speech disturbances. When these are remembered, localizing diagnosis in the majority of cases is not difficult.

Type I.—*Motor aphasia,* the inability to use spoken speech; or the improper use of words, speech-jargon—motor paraphasia.

Type II.—*Word deafness,* total or partial auditory aphasia; sensory paraphasia—the inability to understand spoken speech.

Type III.—*Word blindness* (alexia), the inability to understand printed or written speech.

In *motor aphasia* the lesion is in the third frontal convolution (Broca's center), or subcortical, in the speech tract which runs from this convolution to the inner capsule.

In *word deafness* the lesion is in the middle or posterior division of the first temporal convolution or its vicinity; in paraphasia there is interference with the bundle connecting the first temporal with the third frontal convolution.

In *word blindness* and its concomitant disturbances the lesion is in the angular gyrus, that is, in the posterior lower parietal convolution.

A description of a fourth type of aphasia, the so-called motor *agraphia,* is still being carried from one textbook into another, but it is purely speculative. Though Charcot defended the existence of a writing center at the foot of the second frontal convolution, no convincing anatomical proof for this contention has ever been furnished.

More important than the preceding one is a type of speech disturbance called *optic aphasia.* This is characterized by the fact that a patient so affected recognizes an object and knows its uses, but cannot name the same without resorting to another sense, as, for instance, that of touch. The lesion is found in the bundle leading from the occipital lobe to the first temporal convolution.

Related to the last, but not identical with it, is the condition called *soul or mind blindness.* By this we mean a patient's inability to recall the significance of a familiar object by sight or any other sense; he sees with his eyes, but not with his brain; and even accustomed locations appear strange to him. The exact localizing importance of soul blindness is not quite certain, but when found in a patient whose intelligence is not impaired it constitutes a pathognomonic sign of bilateral occipital lobe disease.

Occipital Lobe.—Tumors growing here produce mainly disturbances of vision. The symptoms are those of either irritation or of paralysis. The former usually appear first in the form of visual hallucinations of colors or figures (men, animals, objects). The hallucinations are mostly unilateral, and appear upon the side opposite to the lesion; for instance, in a lesion of the right occipital lobe symptoms will be on the left side. Sooner or later homonymous hemianopsia without Wernicke's hemianopic pupillary reaction is added to the hallucinations.

To accurately localize a lesion therein one must recall the physiologic data of the occipital lobe.

The visual center, according to modern physiologists—and of clinicians Henschen adopts this view entirely—is limited to the calcarine fissure; the upper portion corresponding to the upper retinal quadrant, while the lower quadrant is represented in the lower portion. The temporal side corresponds to fibers coming from the same side of the retina, the nasal side representing those from the opposite half of each eye. There are, then, crossed as well as uncrossed visual fibers. The occipital cortex elaborates both light and color perception. It is assumed that the macular vision field is bilaterally represented; not so the peripheral field. It is sometimes possible, by carefully measuring the fields of vision during the growth of a tumor, to determine the situation, as well as the extent of an occipital lobe tumor. Blindness in any part of the visual field can only be produced by a lesion of the visual centers, or fibers which lead to them, while irritation of the still functionating occipital lobe, and especially of its lateral cortex, produces only visual hallucinations.

It may be definitely stated that, in the absence of visual disturbances of any kind, no tumor is likely to be found in the occipital lobe.

Basal Tumors.—The principal tumors observed at the base of the brain either spring from the hypophysis or belong to the gummatous variety of syphilis. In either case vision is disturbed because of direct involvement of the visual paths. Here aneurysm, sarcoma, and carcinoma may also be found growing from vessels, periosteum, or bones.

Basal tumors situated anteriorly to the pons may cause pressure upon a cerebral peduncle and a third nerve, producing oculomotor paralysis of the same side and paresis or paralysis of the extremities on the opposite side—the crossed hemiplegia of Weber's syndrome. The tumor may extend transversely and cause a similar paralysis on the opposite side. Extending still more posteriorly, pressure may be exerted upon the trigeminus nerve or the Gasserian ganglion, producing either neuralgia or anesthesia in the distribution of the fifth nerve.

Tumor in the neighborhood of the *optic chiasm* not only affects the chiasm itself, but may also involve the surrounding structures.

Following are some of the most important symptoms caused by neoplasm in this vicinity:

1. In tumor of one optic tract blindness may appear in a quadrant of the visual field at first, to be followed later by complete homonymous hemianopsia with hemianopic pupillary inactivity. When the tumor extends toward the cerebral peduncle, there may be hemiparesis on the same side as the blind visual field and, in addition, paresis of the oculomotor nerve upon that side. In those rare instances of pressure upon the gyrus hippocampus or the olfactory bulb there may develop in addition olfactory disorders—either hallucinations or loss of smell.

2. The tumor, usually a gumma, begins in the optic chiasm; visual disturbances appear with irregular hemianopic defects at first, to be followed by bitemporal hemianopsia, eventuating in complete blindness. Instead, there may be optic neuritis or optic atrophy with ocular palsies; when the olfactory structures become implicated in the growth, anosmia may be added.

3. The most characteristic symptom-complex of a tumor in the region of the optic chiasm consists of bitemporal hemianopsia with hemianopic pupillary reaction, anosmia, and oculomotor palsies.

4. *Hypophysis Tumors.*—The neighborhood symptoms are identical with those of other tumors about the chiasm. There is at first blindness in the outer fields of vision—bitemporal hemianopsia—later complete amaurosis. Optic neuritis and anosmia are rare, but may occur. In some cases visual disturbance is the only symptom present. With tumor in the hypophyseal region is frequently associated the condition of acromegaly. This disease is characterized by enlargement of the bones of the hands, feet, and of the head. A radiogram, which should be made in every case, will almost always demonstrate the presence of enlargement of the sella turcica.

Cushing distinguishes three sets of symptoms in hypophyseal tumor: (1) neighborhood symptoms, (2) general pressure symptoms, (3) symptoms that concern the gland itself.

The *neighborhood symptoms,* visual and ocular disturbances, have already been described.

General pressure symptoms are those of other forms of brain tumor. In their evolution hypophyseal growths break their dural covering, grow upward, and produce directly pressure symptoms, or, indirectly, by obstructing the foramina of Monroe, thereby creating a ventricular hydrocephalus.

As regards the *glandular symptoms,* we have (1) the striking picture of acromegaly, with the enlarged extremities, which is unmistakable; (2) the Fröhlich syndrome, which is characterized by a peculiar tendency to obesity with sexual underdevelopment or infantilism.

The cause of acromegaly is generally held to be due to a hyperplasia or adenoma of the anterior part of the hypophysis, the glandular portion. Fröhlich's syndrome, on the other hand, is thought to be due to an atrophy of the gland, which may be produced by the pressure of a tumor in its vicinity. If this be the true pathology, it favors the view that the symptoms of Fröhlich's syndrome are caused by a diminution in the glandular secretion—*hypopituitarism;* while acromegaly is assumed to be caused by the very opposite, namely, hypersecretion—*hyperpituitarism.*

Other symptoms caused by neoplasm situated at the base of the brain are polyuria, diabetes, amenorrhea, and impotence, which are attributed by Cushing to metabolic disturbances in the glandular portion of the hypophysis.

THE MEDULLA OBLONGATA.—Tumors here produce complex pictures, owing to the number of structures crowded together into a small space. The principal symptoms indicate nerve involvement from the eighth to the twelfth inclusive. There may be Ménière's syndrome, or palsies of the palate, vocal cords, and tongue, with or without paralysis of the upper and lower extremities. By pressure upon the vital centers in the medulla cardiac and respiratory disturbances are produced.

CEREBELLUM.—Cerebellar growths produce both general and local symptoms. The last are caused in small part only by pressure upon, or destruction of, the cerebellum itself; the greater number of symptoms are produced by involvement of neighboring structures, such as pons, crura cerebri, and medulla oblongata.

The principal *general symptoms* are: rapidly developing optic neuritis, headache, vomiting, and nystagmus.

Local Symptoms.—The cerebellum, though neither the seat of intelligence nor of the special senses, exercises a regulating control over the neuromuscular sthenic, tonic, and static functions. By its coördinating mechanism it regulates all body movements. The symptoms caused by destruction of the cerebellar substance itself are asthenia, atonia, and ataxia.

In a tumor of the cerebellum there are, consequently, (1) weakness—asthenia—especially of the muscles in the lower extremity on the same side; (2) disorder of equilibration upon standing, which disappears in the recumbent posture; (3) swaying upon standing and walking—cerebellar ataxia; finally, pressure upon the vestibular nerve causes the early and important symptom, (4) vertigo. All of these symptoms belong exclusively to tumor of the vermis, while neoplasm of the hemispheres may give no manifestations until it encroaches upon the former.

Concerning the exact localizing diagnosis, which is of so much importance to the surgeon, this cannot be made from cerebellar symptoms alone. For this we must also invoke the aid received from pressure symptoms upon neighboring parts, as the acoustic and facial nerves and the pyramidal tracts. After inquiry into the exact order in which individual symptoms have appeared, it may be possible to decide whether the tumor is situated ventrally or dorsally, superiorly or inferiorly. Tumors growing laterally will most probably press upon the cranial nerves from the fifth to the twelfth inclusive.

Though in many cases cerebellar tumors have been found as localized, surgeons have repeatedly failed to find them, because they were not correctly diagnosed.

CEREBELLOPONTINE RECESS.—In this variety of tumor the fifth to the twelfth cranial nerves on one side are usually affected. Especially characteristic for this location are the so-called acousticus tumors, springing from the acoustic sheath. Pontile angle tumors are differentiated from other neoplasms by their unilateral beginning, usually in the cochlear and vestibular nerves; later the fifth, seventh, and ninth nerves are attacked. The affected motor cranial nerves show reaction of degeneration and loss of reflexes. Added to the other symptoms, there may be the usual cerebellar syndrome of ataxia—the uncertainty in standing and walking—and crossed or unilateral paresis in the extremities, because of pressure upon the pyramidal tracts.

DETAILS IN LOCALIZING A TUMOR.—Before deciding upon surgical treatment, a focal diagnosis should be made. For this a painstaking inquiry should be undertaken into everything pertaining to a possible cranial injury. Further, a complete physical examination should be undertaken, not only of the entire organism, but particularly of the cranium.

Inspection.—Rarely will one discover a tumor upon inspection of the skull. This is possible only in the growths of dura or skull pushing their way outward. Skull deformities may be observed in children, especially in connection with echinococcus or other large tumors. An edema of the scalp and enlargement of the superficial veins may also betray the existence of an underlying tumor.

Palpation.—When the skull has become very much attenuated, the palpating finger may experience a peculiar parchment-like, crackling sensation. The hand may discover slight edema and a difference of temperature on the affected side; perhaps also some tenderness.

Percussion.—Tenderness elicited upon percussion is of greater value than when obtained by palpation, provided the soft parts are not painful to the touch. In rare instances it was possible to determine the existence of tenderness at the base of the brain by palpating the pharynx. When tympanitic percussion sound, or so-called cracked-pot resonance, is present, it usually indicates a tumor underneath; in children it may mean hydrocephalus.

Auscultation.—Blowing noises are occasionally heard in aneurysm and vascular growths; these are probably caused by narrowing and compression of the large vessels at the base. In small children with open fontanelles this is a normal phenomenon.

Depth of Tumor.—The next question to decide is: *How deep is the tumor?* This can be answered by determining whether the tumor originated in bone, dura, pia, or brain substance. As deep-seated must be classed all basal tumors, also those of the pons and its vicinity, tumors of the medulla, the ventricles, the central ganglia, and of the adjoining white substance. The cortical and subcortical neoplasms of the entire convexity are classed as superficial tumors.

A. Characteristic for the beginning of neoplasm from the skull are: (a) subjective symptoms, such as severe superficial pain, strongly localized, usually in conjunction with local tenderness upon pressure and percussion; (b) objective changes: edema of the scalp, injection of the veins of skin and skull; (c) external swelling or deformity, such as indentations and scars; (d) changes in the surface temperature; (e) a history of fracture or other trauma.

B. Tumors having their origin in the *dura* are: (1) hematoma, in which the etiology of alcohol, psychosis (general paresis), and sometimes trauma or syphilis can be ascertained; (2) fibromata are slow-growing tumors with symptoms of low intensity; (3) syphilis is accompanied by violent nocturnal headache and has a tendency to invade the psychomotor and speech centers, causing Jacksonian epilepsy, paralysis, and aphasia.

C. *Pial tumors* resemble those just described and occupy a similar position surgically. Aside from troublesome headaches, they are like cortical tumors. The most common forms are: fibromata, gummata, tubercle, fibrosarcomata.

D. *Cortical tumors* produce first irritation, later paralysis. The order in which symptoms appear is helpful in making an exact localizing diagnosis. In the order of importance (a) motor irritation phenomena head the list. In cortical tumor spasm precedes paralysis, while in subcortical neoplasm the opposite usually takes place. This is not an invariable rule; muscular twitchings may rarely precede paralysis even in subcortical growths.

(b) Sensory symptoms. Paresthetic disturbances precede the anesthesias.

(c) Special senses. Hallucinations of the special senses are also indicative of cortical tumor. Great importance is attached to unilateral auditory hallucinations, but it must be determined that these are caused by irritation of the auditory centers, and do not originate in the inner ear. When the occipital lobe is the seat of irritation, unilateral visual hallucinations may appear even in a blind visual field or in connection with developing blindness.

(d) Aphasia and psychic irritative phenomena, such as moodiness, depression, apathy, etc., point to a cortical situation of the tumor.

In addition the following points argue for a superficial situation: (a) Headache early in the course, with more or less constant localization of the same over a certain point; (b) localized tenderness on percussion (not an infallible sign); (c) bulging of the cranium or signs of past injury, such as depressed scar, etc.; (d) a sign of negative value is the absence of optic neuritis; this has been noted in a large number of tumors situated over the convexity of the brain.

Cortical tumor cannot be excluded, even when the symptoms previously detailed are absent, for the reason that slowly growing benign tumors often run their course without irritative symptoms, with very little headache, and only slight tenderness over their location (Henschen).

Prognosis.—The prognosis of cerebral tumor without operation is unqualifiedly bad, excepting, perhaps, the cases of gummata, which respond to antiluetic treatment.

It has long been known that tumors may undergo regressive changes, becoming converted into fat and calcareous products. Occasionally glioma and sarcoma may liquefy, become transformed into cysts, and thereby lose their tendency to compress healthy brain tissue. To what extent a tumor may become arrested in its development, or so reduced in volume that no more symptoms are produced, we cannot foretell with any degree of certainty. Byron Bramwell reported a case in which a cerebellar tumor became encapsulated and in course of time ceased to produce symptoms.

Prophylaxis

Only with reference to syphilis can we speak of tumor prophylaxis. To what degree proper antiluetic treatment can prevent the appearance of gummata is not certain, but we believe that it may do so. Remembering that head trauma often constitutes an exciting cause for the development of gumma, injuries occurring in an individual who has had syphilis demand most energetic antispecific treatment.

In respect to tuberculous neoplasm, trauma must likewise be avoided, as children with tuberculous tendencies are apt to develop tuberculomata after an injury. From the prophylactic viewpoint especial attention must also be paid to existing chronic inflammations of the nasopharyngeal mucous membranes.

Regarding the interval of time between the receipt of an injury and the development of a tumor, various observers make different statements —from "a short time after trauma" to several years. In many of the operated cases the data in respect to a trauma having preceded tumor must be considered uncertain. It is, nevertheless, probable that in some cases there is a clear connection between trauma and tumor, especially when the latter develops under the scar caused by an injury.

Treatment

As soon as the diagnosis of brain tumor is made and corroborated by careful neurologic examination, active treatment must begin. The therapy will be: (a) hygienic, (b) by means of internal medication, (c) by external remedies, (d) surgery.

Hygienic Treatment.—The general *hygienic* treatment consists in preventing the slightest degree of trauma and congestion to the brain. The patient's life should be so regulated as to avoid all mental friction and shock. If a child, attendance at school should cease as soon as a diagnosis has been made. Adults should likewise abandon serious study, and should endeavor to live free from mental stress.

The diet should be light and may consist principally of milk and soups. Large proteid meals and foods causing cerebral congestion must be avoided. Alcoholic drinks and coffee are to be entirely prohibited. Especial attention must be given to regular daily evacuations of the intestinal contents. Moderate outdoor exercise is permitted, but the patient must never be left alone, for apoplectic attacks and vertigo or epilepsy may occur when least expected.

Symptomatic Treatment.—The most troublesome symptoms are headache and insomnia; both are probably caused by either increased intracranial pressure or by direct irritation or destruction of tissue.

Insomnia not infrequently appears early and reaches a high degree, especially when there is also severe cephalalgia. Treatment aims to reduce the general nervous irritability and to produce sleep directly. The first object is best attained through the administration of sedatives, as sodium bromid in 15-30-grain (1-2-gm.) doses three to six times daily, with a double dose at night if necessary. To produce sleep directly 7-15 grains (0.5-1 gm.) of veronal or 15-30 grains (1-2 gm.) of chloral may be tried. Of other remedies sulphonal must be mentioned, which can be ordered in doses of 15-30 grains (1-2 gm.) at night, but not for long periods. Antipyrin and aspirin have also been used successfully for this purpose. In obstinate cases of insomnia powdered extract of opium in nightly doses of 1 grain (0.06 gm.) are given. In the most obstinate cases it may be necessary to inject hypodermically one-sixth to one-third grain (0.01-0.02 gm.) of morphia sulphate in order to produce sleep.

Headache is undoubtedly the most serious symptom from the patient's viewpoint; it may continue for several years and may make his life unbearable. Not rarely with increasing stupor the headache becomes more intense. For the most part the head pain is constant, although it may appear in paroxysms. To combat it we employ general hygienic remedies, which aim to prevent congestion. Of the nerve sedatives, sodium and potassium bromid occupy the first place; they may be given for a long time; in fact, throughout the entire duration of the disease, in doses ranging from 45-90 grains (3-6 gm.) daily.

The remedy enjoying greatest popularity is potassium iodid. According to most authorities, notably Horsley, Wernicke, and others, iodid of potassium affects favorably all forms of brain tumor headache. For this purpose it is best given in moderately large and continuous doses. Of course it is the remedy par excellence for the headaches resulting from syphilitic tumor, for which heroic doses, up to 600 grains (40 gm.) daily, are administered. Paroxysms of headache require rest and quiet in a dark room. The application of wet cloths wrung out of cold water, or preferably ice placed upon the head, may act efficaciously. Laxatives may occasionally relieve an attack; cooling enemas, as well as cold foot-baths and cool general baths, may also be serviceable. Local blood-letting has sometimes done wonders in these cases. Good results have been seen from the application of wet cups and leeches to the neck. Convulsions, stupor, and somnolence have thereby been quickly relieved. In tumors of the frontal lobe, or those localized at the base of the brain, leeches may be applied to the inner canthus of the eye, the temporals, or the mastoid processes. In anemic patients dry cups are given preference to wet cups.

Vertigo is combated in the same way and requires a sedative regimen and rest in bed.

Vomiting, produced by similar causes as bring on the attacks of cephalalgia, must be treated in like manner. Chopped ice, dropped into effervescent water and administered in small and repeated doses, has proved grateful. In obstinate cases small doses of morphin, codeïn, or cocain may be tried. When cerebellar tumors are the underlying cause, ice to the neck and wet cups over the mastoids are indicated.

Spasmodic twitchings in the hand, arm, leg, and general epileptiform convulsions may become very troublesome. These are treated symptomatically, the same as though they were produced by causes other than neoplasm. When the convulsive attacks become more or less chronic, potassium bromid in combination with potassium iodid, in average doses (15 grains), should be administered. For serial convulsions, which may become life-threatening, nothing is better than morphin injections repeated until relief is obtained, for bromids will accomplish but little in these cases. At the same time it is well to apply ice to the head and to administer chloral enemas. Inhalations of chloroform, frequently repeated, have sometimes succeeded in checking convulsions. More powerful in their effects upon epileptiform attacks are blood-letting and venesection. These measures have not only checked convulsions, but have relieved other annoying tumor symptoms. We must never forget, however, that in advanced cases of tumor the brain is often bloodless from pressure, and that an additional sanguineous loss may produce sudden death by anemia of the medullary centers. Blanched face and general debility, cardiac irregularity and weakness, and particularly advanced age, are all contraindications to venesection and wet cupping. It is remarkable that, after blood-letting, paralysis and optic neuritis may temporarily subside. However, when vision is threatened it is not safe to wait. Operation is then to be resorted to without delay, either for decompression or for radical removal, as vision once lost cannot be restored. This advice is concurred in by leading surgeons and neurologists.

Operation.—The only rational method of treating brain tumor is by surgical operation. As the trephine is a two-edged weapon, it should be employed only after carefully considering the following points:

1. Some tumors spontaneously undergo regressive changes, or become quiescent.
2. Slow-growing tumors may produce very insignificant symptoms, or patients may remain free from symptoms for many years.
3. Syphilitic tumors may disappear entirely after continuous or interrupted courses of antispecific treatment.

An operation should be performed when there is the prospect of either prolonging the patient's life or of making it more comfortable. When neither can be hoped for, operation should be desisted from.

I cannot agree with those who claim that the prognosis as to life is better in patients who have not been operated upon and that brain operations should be abandoned. Regardless of the case citations, according to which patients have lived up to 45 years after the beginning of tumor symptoms, the fact stands out prominently that the great majority of cases die when left alone. It is true that in many instances life has been shortened by the risks incident to operation, but it is equally true that many other lives have been prolonged.

The causes for failure of operation are many. Of prime significance is the fact that it is not always possible to make a correct focal diagnosis. In numerous cases the literature makes record of the fact that the surgeon opened the skull and, not seeing any evidences of tumor, closed up the wound without having opened the dura. A post mortem subsequently revealed a tumor under the operation wound. In other cases a tumor was not discovered at operation, because the surgeon failed to explore the immediate vicinity, but the tumor was subsequently found. Even when symptoms have pointed definitely to the existence of a tumor in a certain location, good observers have found themselves facing a so-called pseudotumor (Nonne and others).

Operability of Tumors.—Which tumors are considered operable? Statistics show that, contrary to expectations, gummata, echinococcus, meningeal tumors, and tuberculomata, when found during operation, were removable, while numerous gliomata and sarcomata were inoperable, either on account of their large size, or because they had infiltrated the neighboring brain substance. The operability of a tumor will also depend on whether it is encapsulated, circumscribed, or diffuse. A circumscribed or encapsulated tumor is usually operable, while one belonging to the diffuse variety is mostly always inoperable. The location of a tumor will also determine whether the same is to be treated surgically or otherwise. While tumors of the convexity are readily accessible, those situated at the base or in the ventricles cannot easily be reached. It must be noted, however, that since 1898 basal tumors have gradually gained favor as objects of surgical attack. Among the basal growths those situated in the cerebellopontile angle and tumors of the hypophysis have been drawn into the domain of surgical therapeutics.

The surgery of the hypophysis has been furthered by a better knowledge of its physiology and of its clinical symptomatology. It is chiefly due tc the daring of men like Horsley, von Eiselsberg, Cushing, Kanavel, Halstead, and a number of others that the hypophysis has become accessible to the surgeon's reach. The operative technique has received such wonderful improvement that hypophyseal tumors can now be reached through extracranial routes, and the customary dangers of cranial operations can thereby be entirely avoided.

Operations upon the occipital, parietal, temporal, and frontal lobes have not been uniformly successful.

The various kinds of operation are: (a) exploratory, (b) palliative, and (c) radical.

Exploratory Operation.—Many authorities consider exploration, in the case of a doubtful focal diagnosis, justifiable in the hands of careful operators. Deaths have occurred within a few hours to a few days following operation. In these cases either no tumor was found or only partial extirpation has been done. Henschen is of the opinion that exploratory operations are not to be undertaken except in the presence of the most serious symptoms, such as impending blindness, etc., and when the patient demands it.

Palliative Operations.—The following are the most useful palliative surgical measures: (1) Lumbar puncture; (2) ventricular puncture; (3) decompressive operation.

(1) *Lumbar puncture* is the easiest and least harmful of any of the palliative operations proposed. It can be done repeatedly and without an anesthetic, but must be executed slowly and with care, as the sudden withdrawal of spinal fluid causes harmful reactions. One great danger is the crowding down of the pons-medulla into the foramen magnum, with the production of almost instantaneous death, especially in tumors of the posterior fossa. A good plan is to withdraw a small quantity of fluid with the patient in the Trendelenburg position. As the withdrawal of fluid through spinal puncture can produce a decided temporary reduction of intracranial pressure, this procedure is recommended in all

those cases in which more serious operations are contraindicated. It has been found useful for the relief of severe and constant headache, vomiting, etc., provided these symptoms are the result of internal hydrocephalus. The puncture may have to be repeated several times, as permanent drainage cannot be introduced in the spine. The amount of fluid to be removed at each sitting varies from 50-60 c. c. In one of my cases of inoperable cerebellar tumor a cautiously performed lumbar puncture relieved a persistent headache almost instantaneously.

(2) *Ventricular puncture* after trephining has been undertaken in a limited number of cases. Reports indicate that there has been relief from pressure symptoms, such as headache and vomiting.

(3) Of greater practical importance is the so-called *palliative operation.* Numerous observations have demonstrated that the mere removal of a portion of the cranial vault in cases in which a tumor was either not found or could not be extirpated has given great relief. Violent headaches, which previously had embittered the patient's existence, had disappeared after the operation. Perhaps the weightiest reason for its performance is to save the patient's vision after choked disc has developed, for, when secondary atrophy supervenes, permanent blindness is inevitable, and the time has passed for any kind of surgery. Decompressive operation is advocated by many competent observers, of which number might be mentioned Oppenheim, Sahli, Bruns, Bramwell, Horsley, Sänger, Cushing, and others. In fact, Cushing has perfected a method of his own, which aims to utilize the strong temporal muscles as a covering for the brain defect made by the removal of bone. He maintains that his operation prevents or at least minimizes the amount of cerebral hernia which follows other forms of decompression. The Cushing decompressive operation, performed over the right temporal area—a silent portion of the brain—has been generally adopted by surgeons in this country. Sänger, of Hamburg, long ago suggested that the right parietal lobe, another silent area, be utilized for decompression. This has become the popular decompressive operation in Europe. Either of these palliative operations may be resorted to when the patient's life is in danger, or when symptoms become unbearable. It must never be lost sight of that such operations are not devoid of danger, and that a number of deaths have followed. Here, as elsewhere, the danger of the operation lies more in the operator than in the operation. For the careful operator the decompressive operation should have no terrors.

Trephining Operation with Extirpation.—The object of this operation is to remove a tumor in whole or in part, with a view of prolonging life or relieving symptoms. As to what degree extirpation has been successful in either respect we still lack exact information, as surgeons are loath to report their failures, and preferably select for publication their favorable cases.

The mortality after partial or total tumor extirpation is still great. The fatal cases mostly belong to the category in which a tumor was not totally removed on account of its large size or because of its being too diffuse. Patients who survived the radical operation were benefited in the larger number of instances, though permanent results were obtained in only about 3 to 4 per cent. of cases. Some patients were so far restored to health that they were able to resume their ordinary activities. In searching through the literature one encounters numerous records of cases in which after extirpation of a tumor life had been prolonged for many years.

Of the various kinds of tumor, operative cures have been frequently observed in gummata. In gliomata and sarcomata recovery after operation was not as frequent; but in a number of instances patients have lived up to one year. The operative prognosis is relatively good in echinococcus disease, and recoveries have been reported after operations for the removal of tuberculomata.

Dangers of Operation.—The most common dangers of operation are: shock, hemorrhage, debility, sepsis, and meningitis.

Among the causes producing death after operation shock occupies the first place; patients have died suddenly with symptoms of cardiac or respiratory paralysis. In cerebellar tumors this is probably due to direct implication by pressure of the vital centers in the medulla; in others pressure may have been from a distance.

Hemorrhage has produced fatalities more often in extirpated than in non-extirpated tumor cases. Death has also been caused by extensive sinus hemorrhage, owing to anomalies of the torcular herophili; or when a tumor originated from the sinus itself. Fatalities from hemorrhage usually occurred before the expiration of two days following operation.

Sepsis and meningitis developing after operation have caused a fatal issue within a few days. When death has followed careless asepsis it took place not later than a month after operation.

With Henschen I must state that a study of the exact causes of death following operation should impress us with the following rules:

(1) Carefully observe strict asepsis and antisepsis, so as to prevent septicemia and meningitis.

(2) Avoid the deleterious effects of hemorrhage by carefully treating the patient before and after operation, so as to increase his powers of resistance.

(3) Minimize the effects of shock by operating in two stages (Macewen and Horsley).

(4) Debilitated individuals and weak children should receive a course of tonic treatment before operation, in order to fortify them for the operative ordeal.

(5) Operate when the tumor is still small and removable; after it has grown to considerable size it belongs to the irremovable kind.

(6) In suspected or frankly syphilitic tumors do not spend more than six weeks' time with antispecific medication. If after the expiration of this period no benefits accrue from large doses of mercury and iodids, and no contraindications are present, the case should be operated at once (Horsley).

(7) In tumors considered inoperable, because of large size, inaccessible position, or because localization is impossible, a decompressive operation may be resorted to with a view of prolonging life.

(8) Cerebellar tumors should be operated in two stages, because of their special tendency to cause respiratory paralysis when the medulla is not given an opportunity to gradually accommodate itself to new conditions of pressure.

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CHAPTER XII

ANEURYSM OF THE CEREBRAL ARTERIES

JULIUS GRINKER

Introduction.—Excepting the small miliary aneurysms giving rise to cerebral apoplexy, aneurysms of the cerebral arteries are more frequent than any other variety.

Etiology.—Most often occurring in connection with the arteries at the base of the brain, the middle cerebral comes first in frequency, then the basilar, vertebral, and, last, the anterior cerebrals. The favorite location for an aneurysm is at arterial bifurcations. We distinguish two varieties: (1) the saccular, or so-called true aneurysm, and (2) aneurysmal dilatation of cerebral vessels. The last variety is found especially in connection with the vertebrals and the basilar artery. Aneurysms vary in size from a bean to a hen's egg. Of the two hemispheres the left is more frequently affected.

Pathology.—Aneurysm of the cerebral vessels has a pathology similar to that of aneurysm elsewhere. Its principal cause is a diseased blood vessel. As the majority of patients are recruited from the young and middle-aged, we assume that the common etiology is cardiac and luetic disorder; only a small proportion of cases occurring in those with senile arterial degeneration. Embolism is also responsible for this condition by producing partial occlusion and subsequent dilatation of the artery caudal to the embolus. Trauma is considered another exciting cause in those who are the subjects of degenerated arteries.

Symptoms.—There may be no symptoms when a slight aneurysm exerts no pressure upon the surrounding brain substance. Besides, an aneurysm may permit a blood current to pass through an affected vessel still adequate to nourish the brain, and will consequently not cause symptoms. When symptoms are present they usually have a gradual beginning and are divided into general and local ones.

Among the *general symptoms* are to be mentioned mental disturbances, such as irritability, forgetfulness, and apathy. Further, optic neuritis and pulsating sensations in the head, occasionally accompanied by a murmur, audible even to the examining physician, are additional symptoms. Pressure upon either the carotid or the vertebrals may cause a cessation of the pulsating murmur, in which event this general symptom may be converted into a local sign of value.

Local Symptoms.—These are similar to those caused by pressure of tumors upon certain portions of the brain and cranial nerves. In aneurysm of the internal carotid artery loss of vision in one eye and paralysis of eye muscles may be produced. When the left middle cerebral artery is involved, symptoms of compression of the under surface of the frontal lobe and the internal capsule may appear. The patient then suffers from a gradually increasing hemiplegia with or without aphasia. In connection with the basilar artery, an aneurysm may cause the usual symptoms of pressure on pons, medulla, and of the cranial nerves springing therefrom—the fifth, seventh, eighth, and the vagus group. In short, we may have cerebral paralysis on one or both sides and of the involved cranial nerves, viz.: severe headache, facial palsy, tinnitus aurium, vertigo, aphonia, dysarthria, dysphagia, and respiratory disturbances.

Differential Diagnosis.—Cerebral aneurysm must be differentiated principally from brain tumor. This may be impossible unless a murmur is heard over the seat of aneurysm. It must be remembered, however, that a superficially placed vascular neoplasm may give the identical symptom. When tumor symptoms point unmistakably to the cavernous sinus, an aneurysm is the probable lesion.

Prognosis.—This is exceedingly grave. It is possible, however, for a cerebral aneurysm to become obliterated the same as other aneurysms—a very rare occurrence. The majority of patients die from hemorrhage by rupture of the aneurysmal sac, or else they succumb to paralysis induced by pressure upon vital centers. When the aneurysm bursts, the patient lapses rapidly into coma, and death supervenes. When a hemorrhage floods the motor centers, convulsions may precede the fatal outcome. Cases do not all terminate rapidly; in some the end is delayed for two to three years from the beginning of a fully developed aneurysm.

Treatment

From the viewpoint of prophylaxis everything must be done to prevent a rise of pressure in the brain. The diet should be non-irritating and consist of bland, nourishing food. Alcohol, tea, and coffee are to be absolutely excluded. Mild laxatives are to be administered daily.

In the majority of cases nothing more can be done for cerebral aneurysm than to attempt to alleviate symptoms. Some maintain that the continuous use of sodium iodid has produced cures. In my own experience this remedy has failed. With a specific etiology, both mercury and iodids are to be pushed to the limit, exactly as though we were dealing with a case of brain syphilis. In some cases of aneurysm of the internal carotid artery the common carotid has been tied with success; in others death was the outcome. Cazin reports a complete cure from digital compression of the common carotid. According to Oppenheim, lumbar puncture is contraindicated, for it has caused instant death by rupturing the aneurysm. A practical point in connection with the surgery of brain tumor is that an operation on a mistaken aneurysmal tumor may cause either rupture and death, or an undue expansion of the sac by the release of pressure, thereby aggravating conditions.

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CHAPTER XIII

THE PARASITES OF THE BRAIN

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Introduction.—The most important parasites infecting the brain are the echinococcus and the cysticercus cellulosæ. Of these the latter are the more frequent. Either of them may be found singly, but in the majority of cases they appear in large numbers diffusely scattered over the brain. Cysticerci follow the soft membranes into the fissures and also invade the ventricles, in which they may float or become attached to the ependymal lining. Echinococci have a similar distribution, but are also found in the medullary substance of the brain.

Symptoms.—The brain has been observed to be literally studded with cysticerci or echinococci, and yet no symptoms were present during the life of the individual to indicate their existence. On the other hand, sudden death has resulted from this disease as the first and only symptom.

The symptoms are vague and variable and are not pathognomonic. Of greatest frequency are convulsive attacks. Most often these have the typical characteristics of hysterical spasms, in that they are of long duration, consist of large movements, and are not accompanied with loss of consciousness, or there may be only clouding of the sensorium. During these attacks the patient may pass through the most grotesque contortions; opisthotonos has frequently been observed. There may be but slight twitchings in certain muscles, as of the face and the anterior portion of the neck. Mere tonic contractions without clonic movements are not rare. In addition there may be the symptom commonly described as globus and also the peculiar sensation of a nail being driven into the head, so-called clavus. In fact, the entire array of symptoms belonging to true hysteria may be found in cases suffering from cysticercus of the brain. On the other hand, the convulsions may appear at irregular intervals and be accompanied by complete loss of consciousness, biting of tongue, frothing of mouth, followed by a dazed condition and somnolence; in other words, the patient may present the picture of true epilepsy, for which the condition has often been mistaken.

Mental disturbances of every grade have been noted in connection with the development of these parasites in the brain. Not rarely imbecility and a state of dementia resembling true general paresis have been seen. Irritability, excitement, delirium, and confusion are the most common psychic manifestations occurring either temporarily or remaining as a more or less permanent condition. Depending upon the location, there may be the focal symptoms of monoplegia, hemiplegia, hemianesthesia, aphasia, etc., which may be of short or long duration. Cysticerci or echinococci situated in the fourth ventricle produce glycosuria, cerebellar ataxia, vomiting, and respiratory and cardiac disturbances. Cerebellar involvement announces itself by occipital headache, vertigo, and a reeling from side to side. Irrespective of the location of the parasite, headache and vertigo are common symptoms. Cranial nerve involvement has its own symptomatology and differs in no particular from that of pressure by a tumor. An important feature of cysticercus disease is the predominance of the irritative symptoms over the paralytic phenomena and the occurrence of intermissions, during which the patient may enjoy perfect health. Perhaps the irregularity and variability of symptoms may be explained by the power of locomotion possessed by the cysticercus.

Diagnosis.—This is very seldom made. One may suspect the disease when there are present cysticerci in the skin or muscles, which can be felt as movable bodies. Excision and microscopic examination of a piece of muscle will make the diagnosis certain. Intermittency of the symptoms may direct attention to the possible existence of this disease. Bruns has described in one of his cases the periodic appearance of headache, vomiting, and vertigo, followed by a relative feeling of well-being. After the attack had passed off, the patient was obliged to avoid rapid turning of the head and sudden changes of position, as these regularly brought on paroxysms of vertigo and nausea sufficiently intense to throw him to the floor. He considered these symptoms characteristic for the existence of floating unattached cysticerci in the fourth ventricle. Oppenheim calls this syndrome Bruns' symptom, but maintains that it also occurs in attached cysticercus as well as in other conditions.

Prognosis.—This is exceedingly grave. However, an arrest of symptoms and even a cure are possible, as was proved by the post mortem finding of calcified cysticerci, which had ceased to produce symptoms during the latter part of a patient's life.

Prophylaxis

Prophylaxis is of the greatest importance. Patients should be warned against the consumption of raw or underdone pork. Those affected with tæniæ should not delay taking the usual tapeworm remedies.

Treatment

The treatment of brain cysticercus is mostly symptomatic. In several cases the motor area was operated on with successful removal of the parasite; but the condition being usually multiple, the futility of such an operation is apparent in the majority of cases. Lumbar puncture here is dangerous, especially when the cysticerci are situated in the posterior fossa. Bruns proposed ventricular puncture in cases of cysticercus of the fourth ventricle, provided the symptoms indicate that the cysticercus is attached. Oppenheim, who first opposed this procedure, became convinced of its efficacy after having witnessed Krause perform the operation successfully on one of his patients. He recommends that patients be prepared for this operation by remaining quietly in bed, with the head fixed in one position, so as to insure permanent fixation of a freely movable cysticercus, if such should be present.

Echinococcus cerebri may run its course without any manifestations. As a rule, the symptoms are those of ordinary brain tumor. Headaches are aggravated by movement, and the patient often has a feeling as though something was moving in his head. It must be noted that, according to Westphal, the swelling has a tendency to extend outwardly, eroding the bones of the skull and even penetrating into the nasal cavity. The bones may become thinned out sufficiently for a fluctuating mass to appear, which can be punctured for diagnostic purposes.

The symptoms usual for tumor in the motor area have given rise to operations which unexpectedly disclosed the existence of echinococcus. As there is no treatment of this disease other than that for brain tumor, the mistake is of no consequence.

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CHAPTER XIV

HYDROCEPHALUS

JULIUS GRINKER

Introduction.—An accumulation of serous fluid may occur in the ventricles or in the subdural space. It may be an acute or a chronic process, congenital or acquired. The accumulation in the ventricles, whether of inflammatory or purely mechanical origin, is called internal hydrocephalus, while a similar collection of fluid in the subdural space is called external hydrocephalus. Acute hydrocephalus is the result of tuberculous and other varieties of meningitis. In this chapter the chronic forms only will be discussed under two subheadings, (1) congenital and (2) acquired hydrocephalus.

1. CONGENITAL HYDROCEPHALUS

Etiology.—The causes of this form are either prenatal disturbances of nutrition or irritation of the meninges, producing an increased secretion of cerebrospinal fluid. Physical and psychic traumata to the mother during pregnancy have also been made responsible for this condition. It is more probable that ill health, drunkenness, and syphilis in the parents are the real causes. There undoubtedly exists a family tendency to this disease, as several hydrocephalic children have been observed in the same families.

Diagnosis.—This is made principally by the changed contour of the skull and the wide separation of the cranial bones, which are late in uniting. With an increase of fluid pressure symptoms likewise increase, either gradually or suddenly. During a sudden accession of symptoms we often have the irritative phenomena of delirium, convulsions, or tremors in arms and legs, nystagmus, and fibrillary twitchings in the face. The patient usually succumbs to an acute exacerbation of the disease. When recovery does occur, the child almost invariably shows retarded development in body and mind, is apathetic, does not walk and talk until an advanced age, and seems to lack the capacity for intellectual growth. Often there is added to this picture optic atrophy with complete blindness.

Prognosis.—Fortunately in many instances the hydrocephalic child does not survive its birth, as brain puncture often becomes necessary to make delivery possible and to save the mother's life. Should a child escape destruction by this means, the prognosis nevertheless remains unfavorable, for hydrocephalus is usually progressive and the patient dies after months or years. In the lighter grades of hydrocephalus a child may live a long time. In the majority of cases, however, death is caused by intercurrent complications or by successive increases of hydrocephalic fluid bringing on a fatal termination. Rarely have patients, usually idiotic, reached the age of 40 or 50 years.

2. ACQUIRED HYDROCEPHALUS

The exact cause of this condition is still unknown. It is possible that a slight congenital hydrocephalus had existed unnoticed for months or years, and, owing to some trauma or other cause, the condition had become aggravated. Henceforth a serious array of symptoms became manifest for the first time—and this may have been considered as the beginning of the disease. It is certain, however, that in numerous cases no such congenital origin need be assumed. There are types which are purely secondary to other diseases, and consequently do not demand special consideration, as they fall under the treatment of the primary conditions causing them. Of this character is the hydrocephalus caused by pressure of a tumor mass upon the venæ magnæ Galeni, when the tumor is situated in the posterior fossa. From like causes the foramina of communication between the aqueduct of Sylvius and the ventricles may become occluded; also those between the latter and the subarachnoid space. It will be seen that a diversity of conditions may give rise to hydrocephalus in one or more ventricles. Possibly many of the convulsive and other attacks occurring in cysticercus disease of the brain are due to the parasites causing similar blockings with ensuing hydrocephalus. In widespread meningeal inflammations of tuberculous or purulent origin the openings of communication between the ventricles themselves, or between these and the subarachnoid space, may be occluded by exudates with resulting hydrocephalus. In addition a number of marantic conditions, as chronic pulmonary tuberculosis, nephritis, diabetes, and cachexias in general, are accompanied or complicated by hydrocephaloid states of the brain.

Diagnosis.—The same diagnostic criteria apply here as in the congenital variety of the disease. In children there will be added to the various symptoms of brain pressure the objective evidences of enlargement of the skull and separation of sutures. In adults objective head signs are usually wanting, but there are symptoms of increased intracranial pressure without focal evidence of other organic disease of the brain, notably tumor, which enable a diagnosis to be made in most cases.

Prognosis.—This does not differ materially from that of the congenital variety. [This statement is not accepted by many authors.—Editor.] There are records of recovery, improvement, and arrest of the disease. Acute exacerbations and a sudden increase of fluid raising the intracranial pressure endanger the patient's life.

TREATMENT OF CONGENITAL AND ACQUIRED HYDROCEPHALUS

As both the congenital and acquired forms of hydrocephalus require similar treatment, what follows will apply to both varieties.

It must be noted that no remarkable innovations have been made in the treatment of hydrocephalus during the past fifty years. The surgical treatment, being now done under better aseptic precautions, has perhaps become somewhat more aggressive, but it is doubtful if it is accompanied by better results. According to Henschen, it would sometimes appear as though the more conservative treatment of the past has been more efficacious.

In every case great insistence must be placed upon the proper hygiene of the hydrocephalic child. A general invigorating treatment according to the best rules of our art should be followed in every instance. These children require plenty of fresh air, sunlight, good food, baths, the care of the skin, and attention to the bowels. It is also essential to prevent as far as possible the occurrence of traumata, such as falls and bruises, which usually come to all children, but particularly to the clumsy hydrocephalic, because of his abnormal brain development.

As regards the causative treatment of the disease, this can seldom be applied, for the reason that in most cases we are ignorant of the underlying etiology. Of course, in hydrocephalus with syphilitic antecedents antispecific treatment is indicated. It would appear as though the results achieved in the past with the iodids and mercury were largely because of a syphilitic etiology. Cures have actually been reported from this treatment, both in the early as well as in the late forms of this disease.

Rachitis has also been thought to play an important rôle in the production of hydrocephalus. In such instances the treatment should be with cod liver oil [phosphorus—Editor], iron, malt extract, fresh air, good food, and hydrotherapy; attention should also be given the digestive and intestinal tracts.

The acute *attacks* frequently occurring in this disease, such as convulsions, stupor, etc., which are mostly due to fresh accumulations of cerebrospinal fluid, require especial consideration. The treatment is very much like that of meningitis. Local blood-letting, antipyretics, salicylates, and cold applications are ordered. When the attack threatens life, either spinal or brain puncture should be performed.

The symptomatic treatment corresponds very nearly with that of the acute form. The main object is to reduce the amount of cerebrospinal fluid and to prevent its reaccumulation. To accomplish this internal and external remedies are applied.

Internally we give first of all the iodids, reputed to be the best absorbent remedy, in the form of the potassium or sodium salts; also iodid of iron. The giving of iodine in one form or another is by no means new, but, as no real progress has been made, we are forced to use the old treatment. In addition mercurials may be tried, a drug formerly much used in hydrocephalus. The most definite indication for its use is furnished by a history of hereditary or acquired syphilis. Calomel in small doses and other laxatives given over long periods of time have also been endorsed in the treatment of this disease. The diuretics digitalis and squills in combination have frequently been tried with apparent benefit. Tonics are always indicated.

External Remedies.—These principally belong to the class of counterirritants, such as the blistering agents, unguentum tartrate of antimony and croton oil, sinapisms, etc. The effects of all these remedies are uncertain, but during the last few years Quincke has again insisted upon their use. Compression of the head has been recommended by many good authorities with the intention of reducing its size and to prevent further enlargement. In some cases the patient is unable to tolerate even slight pressure; in others decided improvement was noted, and even cures have been recorded.

Brain Puncture.—Brain puncture, an ancient procedure, has recently been revived and fervently advocated. While this is really only a palliative remedy, it occasionally relieves the pathological condition present. In this respect its effects are similar to those of tapping for a pleuritic exudate—slight release of pressure seems to start the work of the absorbents. Some patients have recovered by a spontaneous bursting of the hydrocephalus and by an escape of fluid through nose, orbit, mouth, or through a fractured frontal bone.

Mode of Procedure.—In puncturing the brain some advise to allow only a small amount of fluid to escape. Still others desire to limit the flow to the constant dribbling obtained through a capillary trocar or a horsehair drain. Another suggestion is to make several small trephine openings, so as to prevent possible septic infection. When large quantities of fluid are suddenly removed, convulsions, stupor, and death may occur—symptoms which are produced either by cerebral circulatory disturbances or by the dislocation of vital parts of the brain. Huguenin recommends the withdrawal of 60 to 100 c. c. at each tapping and advocates a repetition, if necessary, but warns against aspiration. Immediately after puncture or drainage appropriate compression of the skull should be made. For this purpose antiseptic bandages are now being used.

Results of Puncture.—In the preantiseptic era fatal results were frequent. Since we have learned to withdraw smaller quantities of fluid under aseptic precautions, septic infection is not common. During the last few years the tendency has been to do simple brain puncture or to combine with it drainage of the ventricles. In addition there was inaugurated the treatment by means of so-called lumbar puncture, with which Quincke's name has become inseparably linked. Single puncture is now the prevailing practice—formerly multiple punctures were made.

With the exception of a solitary case here and there, the entire practice of brain and lumbar puncture has yielded meager results. It is, therefore, opportune to give W. Kausch's sanguine views on the treatment of hydrocephalus by ventricular puncture, with an abstract of what is considered his own technique:

He insists first of all upon strict asepsis in ventricular puncture and advises that this little operation be performed by a surgeon. Under all circumstances does he discourage ambulatory treatment. For this operation he selects an open area which leads in the direction of the ventricles, avoiding the motor and speech centers—this in the open skull. For the closed skull he recommends that the frontal region be utilized, carefully avoiding the important brain centers and the larger vessels. He prefers this location to the usual area above the auditory meatus recommended by the majority of writers, and he drains the ventricles by alternately selecting different spots over perfect skin areas. In addition he draws the skin tight, in order to bury the puncture spot and thus prevent infection. In explanation of the effects of ventricular puncture he gives the following lucid statement: "The communications between the ventricles and the subdural and subarachnoid spaces are numerous; likewise between the latter and the venous and lymph channels. The avenues of escape toward the cranial periphery are limited. During the development of hydrocephalus the production of liquor has been so rapid that the outgoing paths could not carry it off; the latter soon became compressed and a vicious circle was established. After the paths had again been made patent and kept so, the hydrocephalus gradually disappeared." After each tapping he measures the pressure, aiming to eventually bring it to below the normal. He does not hesitate to reduce the circumference of the fontanelle 20 cm. after each tapping. Further, he places no limit upon the amount or fluid to be withdrawn; his rule is, the larger the head the more fluid should be removed—from 100 to 300 c. c. have been taken out at one sitting. This procedure he repeats whenever necessary. His only criterion for repetition is the rapidity with which fluid reaccumulates and the fontanelles appear to bulge out. In the beginning of treatment he punctures daily, later once every few days. He believes himself to be the first to have recommended such energetic and persistent puncturing for hydrocephalus. Briefly he attempts to answer a number of objections usually made against his procedure. One objection commonly urged against ventricular puncture and the withdrawal of large quantities of fluid is the great loss of albumin. The author thinks that the loss of albumin (one per cent. in the cerebrospinal fluid), which would amount to about 10 gm. after each tapping, can be easily made up by the administration of proteids. As soon as the accumulation of fluid ceases to be large, the loss of albumin becomes less. Regarding the danger of infection, he believes it to be insignificant when proper precautions are taken. Authors express a fear of many punctures, because of the supposed damage to the brain. His answer is that in the many cases which he has tapped he has not yet seen an injury. Finally, he replies to the criticism that he is creating a negative pressure by contending that this is rather beneficial, as all closed lumina are thereby opened up and are thus prepared to take up the fluid so soon as the pressure rises. He attributes the failures of his critics to infrequent and insufficient tappings. The directions for the treatment of hydrocephalus in children he summarizes thus:

"(1) In open skulls ventricular puncture should be done from the existing open spaces.

"(2) The actual pressure should be measured at the beginning and at the end of a puncture.

"(3) In serious cases withdraw during the first tapping about 100 c. c. of fluid, so that the pressure is lowered by about 20 c. c. water, but do not go below 5 c. c. The next tapping may reduce the pressure to zero, and still later tappings may bring the pressure down to minus, provided no unpleasant results are produced.

"(4) Punctures should be repeated so soon as positive pressures are suspected; if necessary, daily; otherwise, after a few days, or until the skull circumference attains normal dimensions.

"(5) In negative pressure, and when the cranial bones stand apart, use compression.

"(6) Lumbar puncture should be done in the mild open skull cases; in the serious cases, only when improvement by ventricular puncture has already taken place, and it has again become necessary to remove large quantities of fluid.

"(7) The more nearly complete the skull closure, the greater precautions must be used to prevent negative pressures.

"(8) In a completely closed skull negative pressures must be altogether prevented. Do not cause a large reduction of pressure in one sitting, but tap frequently and withdraw small quantities at a time. If nothing can be accomplished in this manner, make a small trephine opening in the frontal region and establish permanent drainage.

"(9) Only after persistent and energetic punctures have failed to relieve should the more complicated operations be resorted to."

The new method of brain puncture certainly deserves further trial under the precautions and directions laid down by its originator.

v. Bókay, on the other hand, claims that with the exception of lumbar puncture surgical methods are insufficient and of no practical importance in the treatment of chronic hydrocephalus. In his hands lumbar puncture according to Quincke has proved eminently successful. He insists that spinal punctures should not be made more frequently than 4-6 weeks apart, and that no more than 50 c. c. of cerebrospinal fluid should be withdrawn at each tapping. This method is applicable only when the foramina between the ventricles and the subarachnoid spaces in brain and cord are not occluded. When this has occurred, he advocates puncture of the lateral ventricles or the so-called "Balkenstich" (puncture of the corpus callosum) described by Anton-Bramann. In cases of intense hydrocephalus even cranial surgery is powerless. He concludes with the assurance that systematic treatment by lumbar puncture can be continued for years without detriment and that the results are best in those who come under treatment early, before long-continued intracranial pressure has done irreparable damage to the brain.

My individual preference is to practice repeated lumbar puncture for a number of months. When the results are either doubtful or symptoms become urgent, I proceed to brain puncture. Only when both methods fail do I consider trephining with the establishment of permanent ventricular drainage.

3. SEROUS MENINGITIS

(*Idiopathic Internal Hydrocephalus*)

Introduction.—This disease may be defined as a low-grade inflammation of the soft membranes, characterized by an edematous exudate into the subarachnoid space and the ependyma of the ventricles. Some have considered the process an ependymitis causing a serous effusion into the ventricles, and have compared it with a serous pleurisy. There are two varieties of the disease, the acute and the chronic type, each differing in symptomatology. Here I shall only describe the acute variety, the chronic type having already been discussed under Hydrocephalus.

Etiology.—Children or young adults are most frequently affected. There is commonly elicited a history of infection, such as typhoid, diphtheria, influenza, pneumonia, scarlet fever, or only tonsillitis, rheumatic sore throat, or plain "cold."

Symptoms.—The symptoms may appear quite suddenly and resemble those of the purulent types of meningitis. The usual onset is more subacute and the symptoms are of less intensity. There are fever, headache, rigidity of the neck, and Kernig's sign. Congestion of the nerve-head is the rule, and even pronounced choked disk is occasionally observed. The fever is not high as a rule, and is subject to great fluctuations; temperatures above 102° are but seldom seen. As a result of brain pressure we have delirium, stupor, and convulsions, all of which symptoms may quickly disappear with the absorption or emptying of the fluid. Palsies of the various cranial nerves may also appear and disappear, depending likewise upon the amount of fluid present.

Prognosis.—Many cases recover spontaneously; others die in spite of treatment. The duration of the disease may last weeks and months, but recovery seems possible at any stage. In a number of cases the acute gradually merges into the chronic form and is then indistinguishable from chronic hydrocephalus.

Differential Diagnosis.—The two conditions for which this disease is constantly mistaken are the ordinary purulent meningitis and brain tumor. A bacteriological examination of the lumbar fluid will differentiate the meningitis forms; but a careful study of symptoms is necessary in order to exclude brain tumor. In fact, one cannot always be certain that both are not present. Every clinician of experience has met cases in which he was unable, for a time at least, to come to a decision as between so-called serous meningitis and brain tumor. For the details of brain tumor diagnosis the reader is referred to the chapter devoted to its discussion.

Treatment

It was Quincke's great merit not only to have discovered a method whereby we are enabled to differentiate the various types of meningitis by merely tapping the spinal fluid and subjecting the same to microscopic examination, but he has also given us a means of treating the disease which he first described. Removal of the cerebrospinal fluid, mostly always under great pressure, is the most efficacious treatment. Unfortunately the fluid but too rapidly reaccumulates. Quincke also recommends the internal administration of mercury and the external application of counterirritants to the scalp. v. Bókay's method, as described under Hydrocephalus, deserves a trial in every case. Essentially the treatment is identical with that given for hydrocephalus.

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CHAPTER XV

SYPHILITIC DISEASES OF THE BRAIN

JULIUS GRINKER

Introduction.—Syphiloma, or so-called gummatous neoplasm, has already been discussed under the caption of Tumors of the Brain. In this chapter we are principally concerned with genuine syphilitic brain affections originating from arteries and membranes.

Pathological Anatomy.—The most frequent type of brain syphilis is the variety called basal gummatous meningitis. In this form the inflammation usually begins in the subarachnoid tissues in the region of the optic chiasm—the interpeduncular space—and extends either toward the anterior or the posterior portion of the brain. The affected membrane has a peculiar spotted appearance, owing to the varying consistency and discoloration of the inflammatory products. The tendency is for the specific process to extend deeply into the fissures and to become intimately connected with the cranial nerves. Particularly the optic and oculomotor nerves become interwoven with the gummatous exudate, are compressed thereby, and appear studded with little swellings. In many cases, however, the cranial nerves are not implicated. It is the large arteries at the base of the brain which show a special tendency to become involved in the gummatous process. The arterial coats become thickened by the infiltration of diseased material, which causes a narrowing of their lumen in spots, the so-called luetic endarteritis.

Apart from the type of diffuse meningitis just described, there are found circumscribed gummatous masses in the neighborhood of one or more cranial nerves. It is also possible for the meningitic process to be localized in one spot, as, for instance, over the oculomotor nerve or the optic chiasm. The characteristic of syphilis of the brain, however, is its tendency to appear simultaneously in several locations. This fact explains the varied symptomatology of brain syphilis. Not infrequently hemorrhages and gummatous swellings are found side by side or in different parts of the brain. Diffuse softening of large brain territories has also been encountered along with the other pathological changes.

The syphilitic deposit must be considered a form of granulation tissue, a so-called granuloma, which caseates in some parts and becomes converted into fibrous material in others. In addition small-celled infiltration is found in the adventitious tissue of the arteries, also in the epineurium, particularly of the optic and oculomotor nerves.

Although not as frequent as at the base, a meningitic inflammation, either diffuse or circumscribed, may be found on the convexity, extending for some distance down toward the base of the brain. Indeed, it is possible for the syphilitic meningo-encephalitic process to cause softening of an entire convolution and even of a complete hemisphere.

A gummatous neuritis, particularly of the optic and oculomotor nerves, has rarely been observed. Sphilitic inflammation of arteries, without pathological changes in any of the other tissues of the brain, may also occur. The arterial thickenings of one or more insignificant branches of the basilar artery belong in this category. Many authorities assume that all forms of localized arteriosclerosis are of luetic origin. Oppenheim considers the finding of circumscribed softenings—not caused by diseased blood vessels—a product of syphilitic encephalitis.

Etiology.—Syphilis of the brain has the same etiology as other forms of constitutional syphilis. During the past few years we have learned more of the true causation of syphilis than in all the previous years combined. In 1903 Metchnikoff and Roux, Neisser and Lassar succeeded in inoculating anthropoid apes with the syphilitic virus, thus enabling us to study experimentally syphilis and its poisons. Shortly after this epoch-making inoculation came the discovery of the *Spirochæta pallida* by Schaudinn and Hoffmann, which made possible exact studies of the character of all syphilitic processes, including congenital lues. Close upon the revelation of the actual cause of syphilis came the wonderful results of painstaking laboratory studies with the cerebrospinal fluid gained from syphilitics by lumbar puncture. Wassermann, Neisser, Bruck, Marie, Levaditi, Plaut, Citron, and others have contributed greatly not only to our theoretical knowledge, but also to the practical clinical diagnosis of syphilis, by the discovery and practical application of a specific serum-reaction, the so-called Wassermann test. It must not be inferred, however, that all our diagnostic difficulties have been removed. Spirochetes are not always found in syphilitic products, nor in all stages of syphilis. In addition the specific reaction of blood and cerebrospinal fluid is not constant, so that a negative test does not mean that syphilis is absent. To still further complicate matters, some of the reactions, cytological and chemical, as well as the positive Wassermann test, are found in the metasyphilitic diseases, tabes and general paresis.

Several observers (Lavallé, Brosius, Nonne, Erb, and others) maintain that there is a distinct form of syphilis affecting the nervous system by preference, which is caused by a so-called nerve-syphilis virus. This view is purely hypothetical.

The various manifestations of brain syphilis appear with greatest frequency within the first two years after infection, gradually becoming less frequent up to the tenth year. At the end of the tenth year after infection active nervous syphilis is rare, while metasyphilitic disorders are common. Exceptionally brain symptoms have appeared during the so-called secondary stage of the disease.

The development of cerebral symptoms in those having had syphilis is hastened by severe trauma, mental and physical stress, emotional causes, and alcoholism.

Symptoms.—Basilar Syphilitic Meningitis.—The symptoms of this type show some uniformity in spite of the many variations which are met with. The patient usually suffers for some time from *headache,* an early and constant symptom of great importance. The headache may appear in paroxysms or is more or less continuous, becoming worse at night. In fact, the aggravated nocturnal cephalalgia is considered almost a pathognomonic sign of syphilitic as opposed to other forms of meningitis. Somewhat later attacks of *vomiting* and *vertigo* occur, and not rarely transient *losses of consciousness* and *general convulsions* appear. The patient's mentality is slightly weakened, showing a moderate degree of dementia; memory defects and general apathy are also commonly present. It is characteristic for syphilis that the stupor is not progressively increasing, as in brain tumor, but that at times the patient can be roused. Further, for hours or days a patient may be in a semi-comatose state, which, upon superficial observation, does not differ from sleep or intoxication. There may be violent emotional outbreaks, attacks of confusion and even mania, alternating with perfect lucidity. Especially remarkable is the change from delirium to coma and the awakening from deep stupor to perfect rationality. With the preceding phenomena, the so-called general cerebral symptoms, paralytic signs may appear, pointing to implication of various cranial nerves.

In conformity with the usual seat of the syphilitic process at the base of the brain, the *optic and ocular nerves,* especially the oculomotor, will become affected. The paralysis or paresis may involve the entire oculomotor distribution or only some of its branches on one or both sides. The abducens and trochlear nerves are more rarely affected, the latter usually on one side. Quite frequently ptosis is the only symptom of third-nerve involvement. In many instances the branch controlling the pupillary phenomena is the only one affected throughout the disease; and even at the termination or recovery of cerebral syphilis pupillary rigidity may remain to tell the story. This internal ophthalmoplegia is rather rare in basal syphilis, but occurs more frequently in syphilitic arterial disease of the brain. In bilateral involvement of the oculomotor nerve one side is usually more affected than the other.

Next to the oculomotor, the *optic nerve* is most frequently the seat of the disease. There may be present an optic neuritis or even typical choked disk, followed by atrophy. Occasionally an ophthalmoscopic examination still yields negative findings, while functional tests already indicate serious trouble. Homonymous or heteronymous hemianopsia, that is, blindness in either the same-named or the opposite halves of the retinæ may appear, to be soon followed by complete or incomplete blindness in both eyes. All of these visual disorders can be explained by the disease-process being localized at or near the optic chiasm at the base of the brain. When the optic nerve itself is the seat of the trouble we may have in addition concentric or irregular narrowing of the visual fields and central scotoma, that is, reduction of central vision.

In case the *olfactory nerve* becomes imbedded in the syphilitic deposit, we may have unilateral or bilateral anosmia, that is, loss of smell in one or both nostrils.

Similarly, the *trigeminus nerve* gives rise to intense neuralgic pains, or the reverse, namely, anesthesia or hyperesthesia in the region of its distribution—depending upon whether the nerve is being irritated or compressed.

When the process extends to the posterior portion of the base of the brain the *seventh* and *eighth* nerves rarely escape. The facial paralysis is of the peripheral type and the auditory nerve affection produces both nerve-deafness and vertigo.

The symptoms of involvement of the nerves springing from *pons* and *medulla* need no detailed description, as they correspond to those produced by non-specific causes.

As has been previously stated, there is no regular order in the appearance of symptoms. With the possible exception of certain forms of tuberculosis of the brain we know of no other condition in which this irregularity and inconstancy are so marked a feature. In fact, the most typical and constant factors in syphilis are atypicity and inconstancy. Take, for instance, the visual symptoms. One day a patient will have normal vision, the next day his visual fields are contracted—the day following his fields are again normal. Likewise, attacks of transient hemianopsia, temporary and recurring blindness, with or without choked disk, are not unusual. And similarly we are not surprised to see fleeting ocular palsies passing through several cycles, that is, they may appear, disappear, and reappear, to again disappear, occur, and recur.

Vascular Type.—From the clinical and therapeutic point of view this variety of brain syphilis is even more important than the preceding one.

The most prominent symptoms occur as the result of occlusion of blood vessels, syphilitic infiltration of the vessel wall favoring clotting within its lumen. Symptoms pointing strongly to arterial thrombosis are hemiplegia and aphasia.

Syphilitic hemiplegia presents features similar to those of hemiplegia from other causes. Paralysis may be either slight and incomplete, or it may be severe and complete. Certain antecedent phenomena of the attack suggest its syphilitic character. First, as regards the *onset.* This is rarely accompanied by loss of consciousness. The patient perhaps notices as the first sign of approaching paralysis a weakness in the leg, followed by loss of strength in the arm and a drooping of the angle of the mouth. Secondly, a symptom frequently encountered in syphilitic thrombosis is more or less *severe headache,* which almost invariably *precedes the hemiplegia* by days or even weeks. But no sooner has the "stroke" occurred than the headache disappears as if by magic, or at least becomes considerably milder. So much have I learned to value the importance of the last symptom that I frequently make a provisional diagnosis of syphilitic thrombosis if I can elicit it in a young or middle-aged individual. This symptom is all the more valuable, as from the therapeutist's point of view the early recognition of syphilis is of immense importance—proper antisyphilitic treatment may prevent irreparable damage to the delicate brain structures.

As in the meningitic variety of brain syphilis, paralysis occurring from vascular disease may be either temporary or permanent. To explain the transient palsies we assume a temporary ischemia or localized anemia of the nerve-centers, while the monoplegias or hemiplegias that have a tendency to become permanent are probably caused by vascular thrombosis. In the last type of cases we may have various sensory warnings, such as tingling and numbness in the extremities about to be paralyzed; in the transient palsies, however, such warnings are rare or do not occur.

Syphilitic endarteritis is capable of producing hemianesthesia, aphasia, cortical hemianopsia, and bulbar paralysis, depending upon the vessel which becomes diseased. The middle cerebral artery and its branches being most frequently affected, hemiplegia and aphasia are common symptoms, but the basilar artery and its tributaries may also be rarely involved. In the latter event we will encounter the symptoms of posterior cranial nerve involvement.

In syphilitic disease of the *convexity* of the brain we have a series of characteristic phenomena pointing to its location. Among these, convulsions, followed by paralysis, indicate the existence of either gumma or inflammation. Localized syphilitic meningitis of the convexity may produce partial or *focal epilepsy.*

If the lesion is on the left side *aphasia* may result, with or without slight attacks of monoplegia or hemiplegia. Aphasia may also be caused by thrombosis of the vessels nourishing the speech centers. There is no distinct type of speech disturbance characteristic for syphilis; motor, sensory and mixed varieties of aphasia may appear as in non-specific cases. It is important to remember that, as in the other symptoms of brain syphilis, variability and atypical character are leading features and aids in diagnosis.

Mental symptoms occupy a prominent place in the symptomatology of meningitis of the convexity. Depending for the most part upon the location of the inflammation in the frontal lobe, the symptoms may present striking resemblances to general paresis, for which it has often been mistaken. This syndrome has been described by writers under the heading of pseudo-paresis and will be discussed under the differential diagnosis of general paresis.

Diagnosis.—In the majority of cases this is not difficult. We must consider: (1) the history or evidence of past or present syphilis; (2) the symptoms themselves—their character, development, and particular grouping; (3) serodiagnosis; (4) the therapeutic test.

(1) A searching inquiry into the patient's *antecedents* will often lead to the desired goal. If no information is obtainable by questioning, the patient's body should be carefully examined for evidences of the disease. Syphilides may still be found, or else pigmented scars, with the parchment-like, cigarette-paper appearance, may obtrude themselves upon the examiner's notice. It is also desirable to look for nodes on skull, sternum, and tibiæ. Fritz Lesser has again emphasized the necessity for examining the base of the tongue for the so-called smooth atrophy, which indicates past syphilis. The finding of perforations in palate or nasal septum and of choroiditis will also be helpful.

(2) The *symptoms* themselves are characterized by a peculiar irregularity and non-conformity to types of organic disease. Symptoms may appear bilaterally, or may change from place to place in quick succession. Certain groupings of symptoms may direct attention to the syphilitic pathology. Thus we may have paralysis of the facial and auditory nerves of one side from periosteal swelling around the internal auditory meatus, or paralysis of the oculomotor nerves and the fifth, from disease near the cavernous sinus or the sphenoidal fissure. There may be paralysis of the bulbar nerves, such as the hypoglossal and spinal accessory, when the disease is a posterior basal meningitis. Transient palsies, localized twitchings and convulsions, with mental symptoms, are fairly characteristic for brain syphilis; also a peculiar state of apathy and drowsiness, alternating perhaps with wakefulness and severe nocturnal headaches. The somnolent condition is peculiar in that the patient may be roused when urged, yet he soon relapses into stupor. This state may last for days, perhaps to disappear and reappear. There are not many diseases other than syphilis in which several organic lesions arise simultaneously in widely different parts of the brain.

(3) *Serodiagnosis.*—The Wassermann test, if positive, will be of considerable assistance in diagnosis when there is doubt regarding the specificity of a certain brain lesion. It must be remembered, however, that the

positive finding does not indicate that a particular brain disturbance is necessarily syphilitic in origin—it merely proves that the patient at some time in his life had acquired the disease. Further, its great limitation for our purpose lies in the fact that it is a general reaction for syphilis and does not attempt to state which organs are affected. A patient may be suffering from a glioma of the brain and also give a positive reaction in his blood, provided he is still syphilitic. As is well known, only the positive Wassermann is of value in diagnosis. Negative findings, therefore, do not exclude the existence of syphilis either in the active or latent form.

The spinal fluid shows an increase of lymphocytes, so-called lymphocytosis, in many cases of cerebral syphilis. Nonne's globulin test will be found positive in a large number of cases; also Noguchi's butyric acid test. As these tests have already been described in the chapter dealing with general syphilis, their mere mention here must suffice.

(4) *The Therapeutic Test.*—When a lesion suspected to be of syphilitic origin rapidly heals under antispecific medication, the presumption is that it was syphilitic, although it must be admitted that some forms of brain tumor, notably gliomata, improve under the same kind of medication, but the improvement is neither so thorough nor so lasting.

Prognosis.—Syphilis of the brain will always be considered the most serious form of the disease. About one half of all cases die within two years of its onset, and one-fourth of all cases recover completely, while the remainder only improve. How long either the cure or the improvement will last nobody can foretell in any case. The outlook is especially doubtful in patients past the age of 40 years.

It will be readily surmised that the more energetic the treatment and the earlier it is begun the better will be the prospects for recovery. According to the experience of those who have seen much of this disease, large gummata and sclerotic forms of syphilis are least amenable to recovery or improvement.

Singling out the various types of the affection, it would appear, according to Hjelman, quoted from Henschen, that cases accompanied by irritative symptoms, such as the epileptic forms and the basilar types of the disease, offer the best prognosis—seventy-one per cent. of the cured cases belong to this group. Hemiplegic forms with psychotic manifestations usually have a grave prognosis. The prospects for recovery in the hemiplegic cases will depend largely upon whether the paralysis is caused by cortical or capsular foci—if by the former the prognosis is more favorable. Syphilitic aphasia, unless due to vascular spasm, or temporary arterial occlusion, is often permanent, especially when accompanied by paralysis. The pathology is usually more or less complete destruction of Broca's center. At this point it may be well to state that the fallacy still prevails in the profession, that all syphilitic affections are amenable to cure, provided one applies proper antisyphilitic remedies. Nothing is further from the truth. When softening of the brain has once occurred, it matters little what caused it, the disease must be considered incurable. Nor can an optic atrophy ever be restored, though it may have been produced by a syphilitic meningitis. The period for action is before complete destruction and atrophy have occurred—only then can we reasonably hope to start the processes of absorption, which, by removal of exudates, relieve the symptoms.

Even after complete recovery has occurred patients are not free from relapses, so-called "neuro-recidives," under which name they have been frequently described by the Germans in connection with the administration of salvarsan injections.

Prophylaxis

The prevention of brain syphilis, to which disease so many young and middle-aged men fall victims, is essentially the same as that of syphilis in general. The ignorance prevailing on matters sexual is alarming; it is surprising how many educated young men unthinkingly throw themselves into the arms of those capable of transmitting this disease. If the laity were more thoroughly instructed regarding the far-reaching consequences of a single infection, brain syphilis would possibly not occur with such alarming frequency.

After syphilis has been acquired the prophylaxis against brain involvement must consist in a most energetic antispecific treatment, carried on consistently for a period of three years. The patient must remain under the physician's observation for at least five years longer, during which time he is to be frequently examined for the development of the first suspicious signs of nervous syphilis. A constantly recurring fact, while examining syphilitic brain cases, is the finding that the majority of them had either received no treatment at all or only an insufficient amount of it during the early periods of the disease. It would exceed the limits of this chapter were I to quote the numerous statistics bearing on this point.

My own experience of over twenty years with a large number of cases of nervous syphilis, both in private and hospital practice, has convinced me of the importance of early and energetic treatment of syphilis, although it must be admitted that occasionally an individual may develop a fulminant type of nervous syphilis while under treatment by a competent physician. These cases are so rare that they constitute notable exceptions. The fact remains that the great majority of cases coming to the neurologist's notice have either not been treated at all, or insufficiently so, and for too short a time, when the first manifestations of syphilis appeared. The fault may not altogether be ascribed to the patients. Perhaps physicians do not sufficiently emphasize the dangers awaiting a syphilitic. Each patient should be told that, although there is no absolute safeguard to prevent the worst forms of syphilis from making their appearance at a later stage, yet the only known measure against such occurrence is radical and prolonged treatment early in the disease. I further believe that patients affected with syphilis should never be discharged as cured until they have been under uninterrupted observation for about five years after all symptoms have disappeared, and when the Wassermann test has repeatedly proven negative in the hands of competent laboratory workers.

Another prophylactic measure of vital importance is the absolute prohibition of marriage to one who has had syphilis. If this command must be broken the individual shall have been entirely free from symptoms for at least five years.

In those syphilitics who are predisposed to nervous disorders strict mental hygiene should constantly be insisted upon. It is well known that nervous syphilis especially attacks individuals who have either inherited a weak nervous system, or who have become debilitated by unhygienic habits. Unfortunately, inherited mischief cannot be remedied—all the more must patients be impressed with the necessity of saving their mental energies by avoiding intellectual overexertion and emotional storms. Not rarely brain syphilis develops after some great psychic perturbation, which evidently had created a point of least resistance. Alcoholism is another factor favoring cerebral lues by the chronic hyperemia which it produces, thereby weakening the cerebral tissues. In the same category belong sexual excesses, insufficient sleep, and improper diet. As gumma of the brain or syphilitic meningitis often develops after severe trauma, and as we are forced to recognize the important rôle that injuries to the head in general play in provoking cerebral lues, syphilitic patients should relinquish occupations exposing them to head trauma of any kind.

Treatment

In every case of already existing brain syphilis energetic antisyphilitic treatment must be instituted without delay. If ever there is an occasion for the immediate application of heroic remedies it is in cases of brain pressure and when important cerebral structures are about to be destroyed. Time lost may here mean death or irreparable damage to the patient and his nervous system. The treatment essentially consists in giving the patient as much mercury and iodids as he can take. Neurologists having to deal with the severest types of syphilis have long been in the habit of combining mercury with the iodids, instead of giving each drug singly. It has been repeatedly found that the administration of iodids alone was inefficacious until mercury was added, and similarly that mercury alone did not act as favorably as when the iodids were also given. The safest and quickest method of getting mercury into the economy is by the intramuscular route.

Of the various preparations of mercury I prefer the bichlorid, injected into the gluteal region, in doses of 1/8 of a grain daily; in fulminant cases even 1/8 of a grain twice daily. Some prefer to inject intramuscularly 1/8 grain (0.008 gm.) of succinamid of mercury daily.

Whichever preparation is decided upon, every precaution must be taken against sepsis, and against perforating blood vessels or depositing the fluid in the region of the large nerve trunks. The skin of the patient, the hands of the physician, the syringe, and the medicine to be injected must be made as aseptic as though an important surgical operation were being performed. The region to be selected is the buttock, the exact spot being the center of a line drawn from the anterior superior spine of the ilium to the upper end of the intergluteal fold, this point being well above and to the outer side of the important vessels and nerves emerging from the pelvis through the great sacrosciatic foramen. Thirty injections of either the bichlorid or succinamid of mercury are considered a course of treatment. During the mercurial treatment scrupulous cleanliness of the mouth must be insisted upon. Teeth and gums are to be energetically cleansed with a 3-5 per cent. solution of chlorate of potassium twice or three times daily. When for some reason injections cannot be given, the next best method of administering the mercury is by means of mercurial inunctions rubbed into the parts nightly, varying the location with each rubbing.

I am in the habit of prescribing a dram to a dram and a half (4-6 gm.) of the ung. hydrarg. 50 per cent. for each dose. My directions to the druggist are to put this quantity into a separate waxed paper. The patient or his nurse is instructed to rub into the parts nightly the contents of one paper, each application lasting about twenty minutes, by which time most of the ointment will have been absorbed by the skin. It is advisable to vary the locality of application as much as possible, in order to prevent irritation of the skin. Particularly must the hairy parts of the body be avoided, since the ointment often causes a dermatitis when used indiscriminately. For this reason I direct that the soft parts of the body be chosen in succession, especially the flexor surfaces —the groins, the bend of the elbows, the popliteal spaces, and the inner surface of the thighs. After each rubbing, a large-sized piece of lint is applied over the part and the patient retires for the night.

Henschen thinks that the mere spreading of a layer of mercurial ointment upon the skin with subsequent application of tight bandages is sufficient to cause the absorption of the drug and saves the disagreeable procedure of rubbing, against which many patients object. A warm bath may be ordered after each series of three rubbings. This treatment is continued for about one month. Simultaneously with the external mercurial treatment, the internal administration of potassium iodid in saturated solution is followed up to the point of tolerance, beginning with 30 drops of a saturated solution three times daily after meals, to be increased each succeeding day with five drops at each administration until about 600 drops daily are being taken.

I am a believer in large doses of potassium or sodium iodid for all forms of nervous syphilis. My experience, which coincides with that of others, has taught me that the larger the dose of iodids the less likelihood is there of iodism developing. It is only in doses of 7-15 grains (0.5-1 gm.) that iodism frequently occurs. In recent years several substitutes for mercury and iodids have been brought forward, and testimonials as to their efficacy have been widely distributed. Personally, I adhere to the old and well-tried remedies. The antispecific treatment as outlined may be given intermittently. A course of "mixed" treatment (mercury and iodids) may be administered for a period of six weeks; this is followed by a period of six weeks of complete rest, during which good food and tonics only are to be given. At the expiration of this time another course of treatment is instituted, which is again followed by a similar rest. During the first year or two after the development of cerebral syphilis my plan is to alternate treatment in the manner indicated. During the intervals of specific treatment the patient is not only given tonics, but is forbidden to exert himself, either mentally or physically. After a series of such courses of treatment, at the expiration of the first or possibly the second year, the patient is urged to take two courses of treatment annually during the next five years. A single course of the mixed treatment each year is then insisted on for the remainder of the patient's life.

Salvarsan Treatment.—A little over a year ago began the era of the new treatment of syphilis by means of an arsenical preparation called "606," and now sold under the trade name salvarsan. As with all new methods of treatment, enthusiasm at first ran too high. With the arrival of some unfavorable reports after the remedy began to be used extensively, many have not only cooled off in their enthusiasm, but some have even gone to the other extreme, namely, they condemn the entire treatment. This is a mistake. Large experience and observation over longer periods of time will undoubtedly teach us more exact indications and contraindications for the use of this powerful and efficacious remedy. Even to-day very few deny the superiority of salvarsan over mercury in fresh cases of general syphilis. Opinions are still divided, however, as to the efficacy of the new drug in nervous syphilis, including the parasyphilitic disorders. Some contend that, following an injection of salvarsan, symptoms have either become aggravated, or else new ones have arisen. Even deaths have resulted from its incautious administration. There are but few physicians who still hesitate to employ this remedy in the early stages of syphilis. It is in the later periods, during the time when nervous syphilis is prevalent, that most physicians will assume a conservative attitude regarding its application. Neurologists and general practitioners will no doubt employ this remedy more frequently when they can be assured that it acts more rapidly and that results are more permanent than with the treatment hitherto followed. Such assurance has not been forthcoming in sufficient strength to warrant one to abandon the known old for the unknown new. Further, we must have more reliable data in reference to the dangers accompanying or following its use in cerebral and spinal lesions. To give the reader an impartial view of the present status of salvarsan treatment in cerebral syphilis it has seemed best to quote in abstract the opinions relative thereto of those who have had large opportunities to test the value of the new drug. I have purposely declined to cite my own experience or to draw conclusions therefrom, as the former is limited and insufficient to form an opinion. Salvarsan was first used on the European continent and it is becoming to permit Continental writers to cite their results and proclaim their views.

On September 20, 1910, at the meeting of the "Deutsche Naturforscher und Aertze," in Koenigsberg, Germany, the entire subject of salvarsan was thoroughly discussed, and it will not be amiss to draw our information from the authorities present at that meeting.

According to Neisser, one of the foremost workers with the new remedy, there is neither immunity nor active and passive immunization, nor is there a distinct serum therapy for syphilis; on the contrary, we depend entirely upon chemical means in the treatment of this affection. As a positive Wassermann test, according to our present views, indicates the existence of active syphilis, although a negative test may only mean that there are too few spirochetes to give the complement-binding reaction, it is our duty to treat syphilis in its early stages in such manner as to kill as many of the spirochetes as possible and to prevent a subsequent reawakening of the few remaining ones. He further advises the use of salvarsan in a suspension of olive oil as less irritating than any of the other methods. He insists that each dose be freshly prepared. The injection is made intramuscularly into the upper and outer border of the gluteal region. He makes repeated injections of the usual dose (0.6 gm.), three to six weeks apart, introducing a course of mercurial treatment during the interval. After outlining his method of treating the manifest symptoms of lues, he pronounces the dictum that a syphilitic must be treated continuously, as in his experience, after the Wassermann has been negative a number of times, recurrences with positive Wassermann are still likely to appear, it being impossible to distinguish the uncured latent cases from those that are permanently cured. Only after many tests have repeatedly proved negative should treatment be discontinued.

Ehrlich himself on the same occasion enumerated what he considered recognized facts in connection with salvarsan therapy: (1) That the specific effect of salvarsan is to kill the spirochetes. When destruction does not take place he thinks the dose was either too small, absorption insufficient, or the spirochetes have become "arsenfest," an expression which indicates essentially that arsenic does not destroy them. (2) The existence of specific antibodies in syphilis was proved by the curative effect upon syphilitic infants of the milk of syphilitic mothers who had previously received salvarsan treatment. As a further proof he cites the beneficial effect upon the specific lesions of syphilitic infants from the serum of patients treated with salvarsan. A single remaining spirochete, he says, is capable of producing a recurrence of symptoms. (3) As regards the effect of salvarsan on the Wassermann reaction this is variously stated by different observers. He explains the phenomenon sometimes observed of a positive reaction appearing after salvarsan, thus: Prior to the injection the Wassermann was negative, because there were too few spirochetes to give the reaction. Immediately after salvarsan has begun to dissolve the few remaining spirochetes and to cause absorption of the endotoxins, the blood will show a positive Wassermann, owing to these metabolic products being taken up by it. Under these conditions this should only be considered a reaction of the organism toward endotoxins and the result of a final cleaning-up. The finding of a positive Wassermann following an injection, when it had been previously negative, may become of importance in differential diagnosis. Any case showing a positive Wassermann after treatment cannot be considered cured. When the positive reaction becomes negative and remains so, either a cure has occurred, or else a transient negative phase has been created by the injection, which killed a number of spirochetes—the rest being incapable of producing a positive reaction. Only after the multiplication of the surviving spirochetes later, which depends upon their number and on the degree of damage inflicted upon them, also on the quantity and quality of the antibodies produced—will a positive Wassermann reaction appear. This must be considered as a recurrence without the external phenomena, which suggests the necessity for additional injections. (4) The so-called Herxheimer reaction Ehrlich regards as a sign of the insufficient action of the remedy upon the spirochetes, because of too small a dose, which does not kill, but merely irritates, and stimulates the production of large quantities of toxins. The rapidity with which certain painful syphilitic affections are favorably influenced by a single injection of the remedy Ehrlich explains as the functional effect of the neutralization by salvarsan on the secretory products of the spirochetes. The time, he thinks, was too short—sometimes only twenty-four hours—to cause anatomical changes. When Ehrlich made this report he had in his possession the records of ten thousand cases treated with salvarsan. He was positive then that not the slightest damage to the eye could be attributed directly to the remedy itself. In a single instance only was death produced by an injection, of which he felt certain that it, the injection, was the sole cause. That accident was probably due to shock, and he expresses the hope that similar occurrences may be prevented by improved methods of administering the remedy. The other fatal cases, about a dozen in all, have occurred exclusively in patients suffering from a serious affection of the nervous system, such as tabes with cystitis and cachexia, bulbar symptoms, extensive cortical softenings, and other similar lesions. In one case of syphilitic disease of the phrenic nerve, of which the anterior roots upon their exit from the upper cervical cord were imbedded in a specific exudate and had become slightly degenerated, the reaction after an injection produced irritation and compression of the already diseased nerve, and the case terminated by sudden respiratory paralysis. Salvarsan should therefore not be used until all the circumstances of a case are carefully considered. When hopeless cases are injected with "606," and the patient subsequently dies, the death must not be attributed to the remedy. On the other hand, even in hopeless cases the remedy has saved lives. In fatal cases it is not the direct poisoning of the nervous system by the remedy which produces the result, but a process analogous to the Herxheimer reaction, with the important difference that here vital centers are affected. He advises not to use salvarsan in advanced cases of general paresis and when disease of the heart or vascular system is present. In one patient who died immediately after an injection a ruptured aneurysm was found upon a post-mortem examination; evidently he would have died shortly without the injection. Removal of the spirochetes by no means cures a serious vascular disease with its fatal consequences.

In speaking of the various modes of administering the remedy Ehrlich says that intravenous injections in doses of 0.6 gm. will be unaccompanied by unpleasant effects in persons of average strength, and usually act quickly. He recommends the combination of intravenous and subcutaneous injections. Treatment is begun with 0.4 to 0.5 gm. intravenously, and after two to four days a subcutaneous or intramuscular injection of the neutral emulsion is given. In nervous syphilis Ehrlich cautions against exceeding the dose of 0.4 gm., as the patients are hypersensitive to the drug and may react unfavorably. Indeed, the smaller doses suffice to kill the few spirochetes present in syphilis of the nervous system.

Wechselmann has obtained remarkable results in a number of cases of choked disk and optic neuritis. He has also tried the remedy in early and advanced optic atrophy in the hope of causing an arrest of the process. The symptoms have never become aggravated as a result of this treatment. In his opinion recent optic neuritis is no contraindication to treatment.

Again taking up the question of salvarsan as a nerve poison, Ehrlich writes in the Berliner klinische Wochenschrift, 1910, substantially as follows: Among the twenty-five to thirty thousand patients who have been treated with salvarsan there was only one case in which a previously healthy eye showed signs of beginning optic atrophy after injection. This patient had been treated with some form of arsenic before receiving "606," which might have produced a hypersensitiveness of the optic nerve. Regarding the reported affections of the vestibular nerve after treatment with "606," they were nothing more nor less than Herxheimer reactions appearing in a latent syphilitic nerve trunk, and not produced by the drug. This nerve is particularly susceptible because of its peculiar situation in a narrow bony canal. Other involvements of the cranial nerves, especially of the acoustic and facial, also of the optic nerve, are not due to the arsenic of the remedy, but to the disease itself. All of these nerve complications occurred in the early stages of syphilis—from the second to the eighth month, a period during which the entire organism is saturated with spirochetes. Besides, the patients had only received a single small injection. In such cases it is advisable to give another injection and to repeat this in three to four weeks, reinforcing the treatment with salvarsan by the administration of mercury during the interval. Wherever this method was followed the results were excellent.

Especially noteworthy are Oppenheim's experiences with salvarsan. Of his ten cases of brain syphilis one case of basal gummatous meningitis showed remarkable improvement after two injections of salvarsan in the dose of 0.3 gm. each. In a second case serious meningeal and pontine symptoms disappeared within four weeks. In two other cases he could not express an opinion because they were too recent, although improvement had already set in. In the fifth case no result was obtained, perhaps because of mistaken diagnosis, the disease probably not being specific. The sixth case, an optic neuritis, showed marked improvement. In three other cases—two with advanced psychic symptoms, hemiplegia and pupillary phenomena; one with syphilitic endarteritis—the remedy proved ineffective. In the tenth case, similar to those that were uninfluenced by treatment, except that there was also aphasia present, there was slight improvement in the subjective and objective symptoms.

Michaelis also reports excellent results with salvarsan in the treatment of syphilitic brain diseases. He publishes instances in which the remedy had a prompt effect and caused the rapid disappearance of symptoms in cases of gummatous cerebral lues with headache, cranial nerve involvement, vomiting, choked disk, and psychic disturbances. The steady improvement he had been able to see from day to day. In six cases of complete choked disk salvarsan had caused their disappearance in all except one, which he had been unable to follow up. In none of the cases had there been unpleasant after-effects. Nevertheless, he advises to begin with a small dose and to increase the same at the second injection. With similar success he had treated a case of syphilis of the pons with clonic convulsions in the muscles of deglutition; also syphilitic hemiplegia upon an endarteritic basis. He calls the treatment of active cerebral lues with salvarsan "a very grateful task," but he also advises its use in old cases of paralysis and contractures, as one can never be certain how much of the pathology is still recent and consequently removable.

It must not be assumed from the sanguine remarks of the authors quoted in abstract that the new treatment has acquired only friends. On the contrary, there are many who think it dangerous to use salvarsan at any time, and strongly urge a return to the old treatment. A series of reports have been published on the so-called neuro-recurrences after the injections, attributable to the drug itself.

Finger, for instance, in speaking of salvarsan in a recent communication, says that it has only symptomatic efficacy, while its by-effects may be disagreeable. In regard to neuro-recurrences he says that, owing to his having been able to keep in view over 75 per cent. of the 500 patients he has treated with salvarsan, he found that forty-four, or nine per cent., presented neuro-recurrences. In other clinics the patients have probably been lost track of before the neuro-recurrences had had time to develop—this he is convinced is the explanation why Wechselmann and others have had only from 0.2 to 1 per cent. of neuro-recurrences in their practice. The frequency and the early development of these nervous troubles render them something entirely new in syphilis. During the same period he encountered only five cases of neuro-recurrence among about 2,000 patients treated with mercury and iodid alone. He adds that when Benario points to the 122 cases of neuro-recurrences under mercury alone he has found in the literature, and contrasts them with the 194 under salvarsan, he forgets to mention in how many thousands and thousands of mercury cases this proportion developed. The percentage is the vital point, and this Benario disregards. Finger has had as many cases of neuro-recurrences among his 500 salvarsan patients as Mauriac encountered among 10,000 syphilitics in the course of thirty years. All the clinical phenomena of the neuro-recurrences seem to be manifestations of syphilis of the brain on the basis of a syphilitic arteritis; that is, a primary vascular affection with secondary involvement of the cranial nerves. All the manifestations of chronic arsenic poisoning are the result of a primary toxic action on the capillaries. The primary injury responsible for the neuro-recurrences is that from the arsenic on the capillaries; the localization of the virus at the points of lesser resistance is the secondary process. Finger recalls that two of his patients, apparently entirely healthy except for recent syphilis, died with brain symptoms not very long after an injection of salvarsan, and necropsy revealed recent tuberculous meningitis, traceable to a softened bronchial lymph-node. The evidence suggests that the salvarsan had set up a destructive process in the tuberculous lymph-node, and this entailed the tuberculous meningitis. This danger was illustrated in a further case in which a child with chronic otitis media also succumbed to fresh suppurative meningitis soon after salvarsan treatment. The tendency to recurrence of lues cerebri is well known, but he has witnessed in several cases neuro-recurrences after the recurrences. Mercury given in conjunction with the salvarsan does not seem to be able to ward off danger, he remarks, and points to three cases of fatal hemorrhagic encephalitis which have been published. In conclusion he says that as neuro-recurrences were observed in four per cent. of his patients with primary and with tertiary syphilis, and in 12 per cent. in the secondary stage, he repudiates salvarsan treatment of syphilis, especially in the early secondary stage. In the same article he says the old assumption as to the importance of vigorous local mercurial treatment has been reinforced, not only for the primary sore, but for all external manifestations at any stage, by the recent discoveries in regard to syphilis, as also the impression that treatment has to be long kept up, until the Wassermann reaction becomes negative at each course of treatment and finally becomes permanently negative.

In an article published in the *Münchener medizinische Wochenschrift* of November 21, 1911, Ehrlich again reviews the entire subject of salvarsan therapy. Of interest to us are the points having a bearing on nervous syphilis. The new treatment, he maintains, is being misjudged by attributing to it every accident occurring in the course of syphilis. Many of the unfavorable results were due to faulty technique. The symptoms of fever, headache, vomiting, and diarrhea, which occasionally follow a salvarsan injection and may persist for several days, have been proven by Wechselmann to be caused by the high bacterial content of the water used in making the solution to be injected. Upon Ehrlich's suggestion Dr. Yakimoff has investigated this point experimentally by introducing into animals affected with trypanosomiasis small quantities of dead bacteria, and he found their usual toxicity increased thereby from ten to thirteen times. It is these fever-producing dead bacteria contained in so-called sterile water used in preparing salvarsan for injection that he holds responsible for the reawakening of old tuberculous processes and also for the development of neuro-recurrences. Possibly similar conditions surrounding the administration of salvarsan in Vienna may explain the great frequency of neuro-recurrences in Finger's clinic. To prevent unnecessary and harmful complications Ehrlich urges that every dose of salvarsan be prepared with freshly distilled water. The after-effects of salvarsan upon the nervous system he divides into two kinds—those which appear soon after an injection, and those noted two weeks to four months thereafter. The early symptoms are the equivalent of the Herxheimer reaction upon the skin, with the difference that here it occurs in nerve trunks situated in narrow bony canals and consequently is accompanied by greater discomfort. Another difference is that in the nerves the reaction takes place much later—24 to 48 hours after injection—which may be accounted for by their poor blood supply, the salvarsan circulating in the blood being slower in reaching them. So soon as this has occurred the swelling and irritation of the nerve may institute a series of functional disorders, commonly of the auditory apparatus. The later reactions are probably caused by the few remaining spirochetes subsequently multiplying in sufficient numbers to produce similar disagreeable symptoms. These are the neuro-recurrences (neurorecidive) so often reported in the literature during the past few months. Ehrlich refers to Benario's statistical studies of the neuro-recurrences, which for him settle the point that the latter are not caused by arsenic. After searching through the literature for a given period, with a view of ascertaining the exact number of neuro-recurrences both from salvarsan and from mercury, he discovered that during the same period there were 123 neuro-recurrences from mercury and 194 from salvarsan. Benario's statistics, made at a time when salvarsan was even more frequently used than mercury, tends to disprove the contention that neuro-recurrences are rare with mercurial medication. Ehrlich thinks Finger's ten per cent. of neuro-recurrences remarkable and as standing in direct contrast with the smaller percentages of other large clinics. He devotes considerable discussion to the causation of the nervous symptoms, and he comes to the conclusion that they are not produced by the arsenic, but by young spirochetes which have become mature after salvarsan had cleared the field. In speaking of the fatalities from the new drug, he reiterates his statements of a year ago, that most cases were either hopeless from the beginning, or that the technique was at fault and the prescribed contraindications for its use had not been followed. In patients who suffer from cardiovascular disease, he advises to begin treatment with doses of 0.1 gm. In looking over the number of deaths from this remedy he remarks that, considering the numerous injections given during the past year (several hundred thousand), there had been fewer accidents from this drug than from chloroform. He warns against the use of large doses in patients showing nervous manifestations early in the disease. In addition he recommends that proper care be exercised during the first few days after an injection. One should be prepared to combat threatening symptoms of cerebral pressure by brain puncture or trephining. He advises as a routine treatment an intensive initial course of salvarsan either alone or in combination with mercury, controlled during the first six to eight months by frequent Wassermann tests. If at the expiration of this period the Wassermann has remained negative, some time is allowed to elapse before a so-called provocative Wassermann is made, which is repeated at the expiration of another six months. This mode of procedure will enable us to differentiate the cured cases from those others requiring still more treatment.

Sufficient has been stated in the foregoing to give the reader a fair résumé of the subject and to serve him as a guide in the administration of salvarsan in cerebral syphilis. I believe for some time to come at least it will be best to adhere to the "mixed treatment" in all forms of nervous syphilis.

Regarding the permanent paralytic disorders resulting from the effects of brain syphilis, they require the same treatment as though they were of non-specific origin. Electricity, massage, and orthopedic measures will come in whenever there are any indications for their use.

With Horsley and others I am of the opinion that, if a syphilitic tumor shows no signs of improvement after a six weeks' course of energetic treatment with mercury and iodids, the further treatment must be surgical, and carried out as though the neoplasm were non-specific. After the removal of luetic growths patients have seemed to respond more readily to the usual antispecific medication.

PROGRESSIVE PARALYSIS OF THE INSANE

(*Dementia Paralytica*)

Etiology.—The most important cause of general paresis is syphilis, either congenital or acquired. With tabes the disease is classed as a metasyphilitic disorder, for there is an appreciable interval of time between infection and the development of symptoms—ten or more years. On account of the absence of existing specific lesions in brain and spinal cord, and because of the inefficacy of antispecific medication, it is thought that the syphilis itself has disappeared, but has left behind toxins. The latter so modify the nutrition of nerve tissues that they become prone to degenerate on small provocation. The strongest proof for the syphilitic etiology has only recently been brought forward by the almost invariable presence of a positive Wassermann in the blood and spinal fluid of the paretic. It would appear that something more than a history of syphilis is required to produce paresis, for many have had syphilis and but few develop general paresis. In the majority of instances the development of the disease was preceded by either chronic alcoholism, mental strain, or trauma to the head.

Pathology.—The cerebral convolutions, especially the frontal lobes, appear atrophied and the membranes are adherent and thickened. Numerous nerve-cells have either disappeared or have become shrunken. There is loss of commissural and of tangential fibers. In many instances similar changes have been observed in the posterior columns of the spinal cord.

Symptoms.—The first evidence of general paresis usually appears in the mental sphere. The patient becomes irritable, unstable; he frequently alternates between depression and exaltation. The intellectual disturbances assume characters varying in different individuals. In many cases forgetfulness is noticed early by the patient's friends or family, particularly when it entails money losses. In one occupying a subordinate station in life mental deficits may for a long time remain unrecognized. There is scarcely a picture of mental disease which has not been reproduced by general paresis—from slight depression to the most violent attack of mania. These episodes are mostly of short duration and differ from the functional psychoses, which they simulate by their grotesqueness and absurdity. In doubtful cases the presence of physical signs enables a correct diagnosis to be made. Occasionally general paresis is ushered in with convulsions indistinguishable from those occurring in ordinary epilepsy. After each attack mental and physical deterioration becomes more marked. If the patient is a skilled mechanic he loses his dexterity and is compelled to abandon his work, although he may still be capable of performing coarse labor. As a general rule the last acquired and least organized accomplishments are the first to disappear.

It will only be necessary to enumerate the most important *physical signs* of general paresis. They are: (1) pupillary inequalities, sluggish or absent light reflex (Argyll-Robertson pupil) in one or both pupils; (2) ocular palsies; (3) slight asymmetry (paresis) of face, tongue, or muscles of palate; (4) weakness in the lower extremities (slight hemiparesis); (5) speech disturbances; (6) disorders of locomotion: spasticity, ataxia, or paralysis; (7) sensory troubles (hypalgesia); (8) exaggeration, inequality, or absence of tendon reflexes; (9) sphincter paralysis; (10) optic atrophy.

The symptoms commonly observed when the patient is brought to the physician are the presence of unequal pupils, which fail to react to light, irregular tremors in lips, tongue, and hands, and peculiar speech disturbances. When the patient attempts to repeat test sentences, as "Round a rugged rock the ragged rascal ran," or "Peter Piper picked a peck of pickled peppers," "truly rural," "National hospital for the paralyzed and epileptics," he either forgets entire words or mispronounces and swallows whole syllables. During conversation his facial musculature, including the tongue, flickers and trembles. There are decided tremor and marked incoördination in hands and fingers when tested in the usual way, as by having patient spread his fingers, or touch his nose with the index finger. The handwriting likewise betrays tremor, incoördination, and general failure of the intellect. The deep reflexes may be entirely absent or unequally exaggerated. For convenience of description the symptoms have been classified into three groups and have been assigned to the three stages of the disease.

(1) The prodromal symptoms, occurring in the incipient stages, are much like those of neurasthenia, except that here there are forgetfulness, lack of correct judgment, and a noticeable deficiency of tact.

(2) The fully developed disease may appear either as exaltation (mania) or marked depression (melancholia). At one time hallucinatory excitement may be the prevailing mood; at another time all these states alternate and a circulatory psychosis may be simulated.

(3) In the final stage the psychic outbreaks subside and a slowly progressive dementia develops, during which the patient is reduced to a vegetative automaton, requiring as much care as an infant.

It must not be forgotten that the division into stages is only schematic. One stage often merges imperceptibly into the other, and long remissions may occur between stages. At any stage the disease may be interrupted by apoplectic attacks, which are characterized by the development of transient hemiplegias and are sometimes accompanied by epileptiform convulsions, rises in temperature and loss of consciousness. The paralytic attacks are usually mild, and the symptoms rarely last more than a few days.

Differential Diagnosis.—In the early stages the disease may be confounded with neurasthenia. In fact, for some little time a definite diagnosis may be impossible, but differences will soon be noted between the simple fatigue symptoms of neurasthenia and the incipient mental deterioration of general paresis. Of course there can be no diagnostic difficulties when the physical signs of the disease appear. But even without these we may recognize the nature of the malady by making detailed searching inquiries. We may learn that the patient himself is not worried about his difficulties, but that his friends are, who bring him to the physician, because they have seen the patient becoming transformed into an irritable, careless, and otherwise strange being. It is quite otherwise with the neurasthenic, who is himself very much concerned about the reason for his illness and asks a thousand and one questions.

Among the several conditions which have to be differentiated from general paresis must be especially mentioned the mental deteriorations of chronic alcoholism and of certain forms of brain syphilis, also non-specific tumors of the frontal lobe.

As against any of the non-luetic diseases we have an excellent means of differentiation in the Wassermann test, but not when brain syphilis is in question. A positive Wassermann indicates that there is either syphilis or general paresis, nothing more. At this point it must be emphasized that while the positive Wassermann does not aid materially in differential diagnosis, for the reasons stated, a *negative* reaction is of considerable assistance in a suspected case of general paresis—it practically excludes it. Wassermann is positive in the blood in from 95 to 100 per cent. of cases, which means that the negative reaction does not absolutely exclude the disease, but makes it extremely improbable. Nonne and Hauptmann have changed all this. Their modified method of using a larger quantity of spinal fluid than has hitherto been the custom enables them to get positive Wassermann reactions in every case of general paresis. We therefore have in our hands a certain means of excluding suspected cases of general paresis. For the differentiation between so-called pseudo-paresis of syphilis and genuine paresis we must still rely upon clinical signs, a subject into which we cannot enter.

Prognosis.—The final outcome of this disease is death within about three years from its beginning. Recently cases have been reported that have lasted eight and ten years, but these are exceptions. Curious remissions occur in this disease, lasting from a few months to one or two years. Dana thinks he has even seen cures; his observations antedated the Wassermann era, and consequently the element of diagnostic uncertainty is present. The tendency is for patients to deteriorate after each epileptiform or apoplectiform convulsion, both in mind and body, until they become bedridden. Many die from intercurrent disorders or from bedsores; some are carried off by infections on the part of the urinary tract.

Treatment

Though general paralysis must be considered an incurable disease, it is not one that does not require treatment. The *prophylaxis* is that of syphilis. The statement "no syphilis, no general paresis," may now be considered proved. Having acquired lues, the best and safest prophylactic against any form of nervous syphilis and parasyphilis is energetic treatment for a long time and a strictly hygienic mode of life, having especial regard for the welfare of the nervous system.

As soon as a diagnosis of general paresis has been made our first endeavor must be to protect the patient's family from financial ruin and the loss of reputation. To this end it will be advisable as early as possible to have a conservator appointed to manage the patient's property and his business affairs. The patient himself must not be permitted to be at large. The best plan is to send him to a public institution or to a closed private sanitarium, where he will be free from all excitement and responsibilities. It is not always possible to convince the relatives that this course is imperative, until it is almost too late to save even a remnant of his worldly belongings. Under an apparent normal exterior it is difficult to believe that the patient lacks the judgment required not to become a prey to all kinds of wild schemes calculated to plunge him into poverty. It is most especially in the early, the so-called incipient stage, that detention is necessary. The quiet and well-regulated life followed in an institution for the insane is conducive to the prevention of the apoplectiform and epileptiform attacks so frequent in the beginning of the disease. At this period, also, outbreaks of violent agitation are common and more easily subdued in a well-equipped asylum. It is quite different during the last stages of the disease; the patient then becomes docile and as manageable as a child, and consequently can be cared for at home without difficulty.

The *direct* treatment of general paresis should include a course of antispecific medication. In spite of the experience that in most cases antiluetic treatment is without the least benefit, I believe each case should be given the benefit of the doubt in the form of a rigid course of mixed antisyphilitic treatment, as one can never be certain how many of the symptoms may be due to a still active syphilitic process. Considering that nothing else is of any avail as regards curative treatment, there should be no hesitancy in employing remedies which are claimed to cure some cases at least.

In respect to the use of *salvarsan* in general paresis opinions are at variance. A number of careful observers have seen remarkable improvement from its use, while others, no less competent, warn emphatically against its employment. Personally I have less hesitancy in recommending this remedy in general paresis than, for instance, in brain syphilis, for in the former disease there is nothing to lose and everything to gain. In the few cases coming within my own observation improvement was noted in the mental symptoms. It is difficult to determine how much of the improvement can be ascribed to treatment, and what amount of it was the result of a spontaneous remission. Most authorities agree that, if salvarsan is used at all in this disease, the smaller doses should be preferred to one single large dose. Neisser recommends as the most harmless and effective method repeated injections of 0.1 gm. of salvarsan.

Referring to the exact mode of administering the mercurial and iodid treatment in general paresis, this will not differ essentially from that recommended under the caption of brain syphilis. The only difference is in the amounts of the iodid, which should be smaller in the metasyphilitic disorder, as patients are more easily affected thereby, there being relative intolerance to the drug in many cases. It is seldom necessary to prescribe more than a dram (4 c. c.) daily of the saturated solution of sodium or potassium iodid. Several courses of the mixed treatment may be repeated, leaving an interval of about a month between each, so as to permit of recuperation from the debilitating effects of the medicines.

Whatever treatment may have been selected—mercurials with iodids, salvarsan alone, or salvarsan alternated with mercury—it is absolutely essential that the patient have rest of body and mind and be freed of all responsibilities. In addition the diet must be simple and nutritious, and no alcoholics, coffee, or tea are to be allowed. The daily routine is to be planned so as to include exercise in the open air, carefully graded hydrotherapeutic measures, and general massage.

The symptomatic treatment of the attacks of *excitement* occurring in the paretic upon slight or no provocation is by prolonged immersion of the patient in a warm bath, a method recently adopted by modern psychiatrists as a substitute for bodily restraint. Only those who have witnessed the barbaric old method of strapping a patient to the bed, or have seen the strait-jacket procedure, can realize what a humanitarian remedy is the continuous bath treatment. In exceptional cases it is necessary to administer small doses of hyoscyamin hydrobromid hypodermically and sodium or potassium bromid by mouth. For the occasional attacks of *depression* nothing has proved more effective than the opium treatment, beginning with a half grain (0.03 gm.) three times daily, to be gradually increased to one and one half grains (0.06-1.00 gm.). The cases of *hallucinatory excitement* can be advantageously treated with a combination of chloral and morphin. In the *hypochondriacal* cases the patients sometimes refuse to eat, because of a delusion that the stomach has become wormeaten or is made of glass. In these instances it is necessary to resort to tube feeding and nutrient enemata.

After the patient has returned to his home, as he may during the last stages of the disease, strict attention must be paid to cleanliness. The bedclothing must be kept perfectly dry and the body cleansed with soap and water; the bony parts are to be well padded with sterile gauze. By exercising the most scrupulous care and constant vigilance it may be possible to prevent bedsores, a complication which often carries off the patient. Another dangerous and almost always fatal complication is the development of hypostatic pneumonia, which can be avoided by frequent changing of the patient from side to side. During this stage it will also be necessary to make daily investigation in reference to urinary retention, which is very common among paretics. By percussion of the abdomen from below upward the bladder may be easily outlined. When this organ has become paralyzed the patient must be regularly catheterized and the bladder washed out with a weak solution of boric acid after each catheterization. The mouth must be thoroughly cleaned several times daily; disregard of this precaution favors the development of a regular bacteriological museum in the patient's oral cavity, slowly poisoning its owner. And last, but not least, attention must be paid to the bowels, as the paretic may go without a movement for weeks, unless compelled by force to submit to a flushing or other means of procuring an evacuation.

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CHAPTER XVI

DISEASES OF THE PONS AND MEDULLA

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1. PROGRESSIVE BULBAR PARALYSIS

(*Progressive Glossopharyngolabial Paralysis*)

Definition.—This disease includes those types of bulbar paralysis in which the course is chronic and the condition characterized by a slowly progressive atrophic paralysis of the muscles of the mouth, palate, tongue, and larynx—or, in other words, in which there is involvement of the cranial nerves from the seventh to the twelfth inclusive. Exceptionally the motor nuclei of the fourth, fifth, and sixth nerves are likewise implicated.

Etiology.—The exact cause of this fatal malady is not known. The following factors are mentioned by writers as provocative of the disease: acute cold, overexertion of the oral muscles, as by playing of brass instruments; physical and psychic trauma, with or without syphilis.

Symptoms.—The disease begins insidiously, although there may be a short prodromal stage, during which the patient complains of pain in the neck and of peculiar sensory discomfort in the throat. Gradually the lips, tongue, and larynx become paralyzed. In the majority of cases the first symptoms are noticed in the tongue, which becomes weak in all its movements; the weakness continues to increase until the organ becomes completely immovable and lies on the floor of the mouth as a lifeless mass. Simultaneously with the tongue paresis speech difficulties make their appearance. At first the letters requiring the coöperation of the tongue, such as *l* and *r*, are imperfectly pronounced; somewhat later, difficulty is also experienced with the enunciation of the labials, viz., *b, p,* and *v.* The tongue, toward the last, becomes atrophied, shriveled, and the seat of numerous fibrillary tremors; upon palpation it has the feel of a soft velvety rag. At about the same time mastication and deglutition are impaired—the patient being unable to eat any but semi-solid foods. When, in the further course of the disease, the lips become atrophic, speech is still more interfered with, and blowing, whistling, as well as the act of laughing, are impossible. Like the tongue, the lips also become paretic and permit saliva to constantly escape from the relaxed mouth. The face at this time has a characteristic appearance. The upper portion, which is unaffected, has an expression of intelligence, while the lower half of the face forms a marked contrast with it—appearing stupid and meaningless. This point is so striking and so typical of the disease that it may well serve as a differential diagnostic sign. Because of the palatal paralysis developing in the evolution of this affection the voice becomes nasal in character and resembles more than a little the speech of post-diphtheritic palsy. There is a further resemblance in the phenomenon that fluids regurgitate through the nares, though for a long time semi-solid food can be taken with impunity. When the pharynx, perhaps slightly paretic at first, later becomes completely paralyzed, food is likely to drop into the larynx and often causes choking spells. In addition, a progressive paralysis of the vocal cords eventually produces aphonia, so that the patient cannot even emit sounds. A still more serious danger is the development of cardiac and pulmonary disorders from involvement of the vagus. The pulse often becomes irregular, and attacks of syncope are frequent. Owing to the patient's inability to cough and expectorate, an ordinary bronchitis may become converted into a pneumonia, thus terminating the case.

Progressive bulbar paralysis, being a peripheral motor neurone affection, the reflexes in the disease-area will be either reduced or abolished. When there is exaggeration of the masseter reflex, or even clonus, we are not dealing with a case of bulbar palsy, but with the beginning of amyotrophic lateral sclerosis. It is important to remember that an electrical examination yields slight degree of reaction of degeneration, although early in the case the normal electrical reactions may for a long time be obtained.

Differential Diagnosis.—In the majority of cases progressive bulbar paralysis is easily diagnosed. A slowly progressive bilateral paralysis of the muscles of the tongue, pharynx, and larynx, without sensory disorders, occurs in no other condition. However, this syndrome may only be the beginning or the end of progressive muscular atrophy, or of amyotrophic lateral sclerosis, in both of which there will be other symptoms to attract attention to the original malady. Similarly, the bulbar symptom-complex may indicate the termination, or a complication, of diseases, like multiple sclerosis, syringomyelia, or tabes.

Pseudobulbar palsy, an upper motor neurone disease, usually the result of two distinct "strokes" of paralysis anywhere above the bulbar nuclei, may give symptoms similar to the peripheral neurone affection. The important points are the history of two separate attacks of hemiplegia, on opposite sides of the body, the presence of the reflexes, and the absence of atrophy and reaction of degeneration.

Asthenic bulbar paralysis—Erb-Goldflam's disease—best known as myasthenia gravis, which was classed until recently under the "functional" diseases, may be mistaken for chronic progressive bulbar paralysis. According to Oppenheim, one of the first to describe the "functional" affection, it is characterized by several points of difference. There are no atrophies and there is present normal electrical excitability of muscles. Further, there is often observed the peculiar electrical change called the "myasthenic reaction," that is, after a few contractions the faradic irritability is exhausted and the muscles cease to contract, but have the ability to recuperate after a short period of rest, to again give normal contractions. The tendency to ready fatiguability is present not only in the muscles supplied from the medulla, but also in those of the trunk and extremities; likewise in the levator of the eye, which subsequently results in ptosis. Perhaps the distinguishing feature between the fatigue of myasthenia gravis and that of bulbar paralysis is that the former ceases after a short period of rest, while the latter persists.

Prognosis.—Progressive bulbar paralysis is a serious disease, which usually terminates fatally in from six months to several years.

Treatment

Although the prognosis is exceedingly grave, something may nevertheless be done in the way of treatment. There are physicians who even maintain that a rationally conducted therapy may cause an arrest of the disease. However, though it is not in our power to prolong life indefinitely, we can make the life that exists more agreeable.

Treatment should be begun early, if possible, in order to ward off the implication of vital centers in the medulla.

A patient suffering from chronic progressive bulbar paralysis must be protected from all harmful influences, such as colds, exertion, and trauma, each of which may have had a share in the production of the disease. Perhaps the most important task in the treatment of this disease is to keep up nutrition, as many a patient dies of inanition by starvation, being unable to feed himself properly. Patients should receive a generous, nutritious diet. As they appear to be capable of swallowing semi-solid food much more readily than liquids, which are regurgitated through the nares, they should be given puddings, soft-boiled eggs, omelettes, and finely-divided meats in liberal quantities. At a late stage of the disease, when the patient is unable to swallow, it becomes necessary to make use of fluid food, and we must be very careful to prevent choking by not permitting the same to pass into the larynx. Occasionally we are compelled to have recourse to nutritive enemas.

A general *hygienic* treatment is equally important. To stimulate the nervous system to greater activity, treatment by hydrotherapy has been advised. The graduated cold water treatment has also become popular. It appears that moderately cool douches applied to the nape of the neck, along the spine, as well as over the face, seem to cause an increased flow of blood to the weak muscles, thereby keeping up their nutrition. Vigorous patients may stand warm baths, while old and decrepit individuals must be warned against them.

Electrical treatment seems to be capable of keeping up the nutrition of the peripheral parts, but I doubt whether it has any effect upon the central nervous structures. Those who believe that central stimulation may do good advise the application of the galvanic current, either to both mastoids, the cervical sympathetic, or one pole on the neck, the other over the pharynx or the face, or the anode may be applied over the neck, the cathode over the angle of the inferior maxilla. For treatment of the peripheral parts, either the galvanic or the faradic current may be used, or galvanism may be alternated with faradism. This may be continued daily for a period of about two months, if the patient's strength permits. Each treatment may last for from ten to fifteen minutes, and the current must be mild, or else more harm than good will result. Galvanism should be applied in the quantity of from ½ to 2 milliampères; faradism only in sufficient strength to see a fairly good contraction of muscles, if no reaction of degeneration is present. The usual effect on the patient is that he feels invigorated; after each treatment the weak muscles seem to functionate better. Temporarily, at least, the patient has no difficulty in swallowing and phonation sometimes improves. After a systematic course of electrical treatment we occasionally notice a marked general improvement. Erb believes electrical treatment should be given a fair trial in every case.

Massage.—The effects are similar to those obtained from electricity. The muscles of the face and of the larynx, as well as the masseters, must be gently kneaded. The combination of massage and electricity has often produced decided improvement in cases not too far advanced. By appropriate treatment a fatal issue may be long delayed.

Medicinal Treatment.—A number of internal remedies have been tried, but not one of them seemed to have exerted any influence upon the progress of the disease. Among those commonly prescribed are nitrate of silver, sodium and potassium iodid, iodid of iron, ergot, arsenic, phosphorus, and quinin sulphate. Gowers recommends hypodermic injections of strychnia nitrate in all atrophic muscular states. Erb, on the other hand, warns against the use of strychnia.

Symptomatic Treatment.—The constant dribbling of saliva may often be reduced to a minimum by the regular administration of atropin sulphate, either hypodermically or by mouth, in doses of one-hundredth grain (0.0006 gm.) three times daily. When cough is troublesome, it may be relieved by means of small doses of opiates or morphin.

Mild attacks of dyspnea are treated by anodynes, combined with atropin. When the attacks become severe and life is threatened by suffocation, tracheotomy is our only means of saving the patient.

2. ACUTE APOPLECTIC BULBAR PARALYSIS

Pathology.—The most common pathological cause underlying the development of acute bulbar paralysis is thrombosis, followed by softening. Very rarely hemorrhages, also emboli from the vertebral and basilar arteries, may produce the bulbar syndrome. Changes in the medulla, similar to those of encephalitis occurring in the cortex and much like the myelitic processes observed in the cord, are not rarely encountered. Indeed, during the recent epidemics of acute anterior poliomyelitis, combinations of acute bulbar and spinal inflammations have occurred.

Etiology.—The following are usually mentioned as causes: trauma, arterial disease, cardiac affections, syphilis, and infections from unknown sources.

Symptoms.—Similar to the chronic form of bulbar paralysis, this disease is characterized by the appearance of bilateral, rarely unilateral, paralysis in the region of the cranial nerves, from the fifth to the twelfth inclusive. The onset is usually acute in the vascular and subacute in the encephalitic cases—differing in this respect from the chronic variety, which is always of slow and insidious appearance. The paralysis involves the muscles of mastication, deglutition, and respiration. Most often the lower extremities are affected in some degree at least. The pathological process being diffuse, the paralysis is neither symmetrical nor limited to the motor nuclei of the medulla, as in the chronic disease. When thrombosis is the cause, paralysis appears rather suddenly—apoplectiform—the same as other vascular attacks in the cerebrum. In the encephalitic variety several days may elapse before the bulbar symptoms develop—the disease setting in, as a rule, with the symptoms of an acute infection, such as headache, chills, and fever. After a few days the bulbar nature of the case becomes evident.

Treatment

The disease is treated along etiological lines. If syphilis is the causative factor, rigid antisyphilitic treatment will suggest itself as the only course open to the patient. The cases due to a vascular accident, as hemorrhage, thrombosis, or embolism, require the same management as prescribed for the corresponding lesions in the cerebrum. Acute encephalitis of pons-medulla is treated according to the directions given for the cerebral disease, of which internal and external antiphlogistics constitute important items. Oppenheim reports considerable success from the administration of large doses of calomel. Above everything else, constant attention must be paid to maintaining the proper nutrition. Patients suffering from paralysis of deglutition may have to be fed through a tube. Precautions must be taken against the development of aspiration pneumonia, a complication which but too often kills the patient.

3. COMPRESSION BULBAR PARALYSIS

Pathology.—Bulbar paralysis may be caused by *tumors* of the vicinity or of its own substance pressing upon the medulla. Similarly *aneurysm* of the basilar and vertebral arteries may produce compression of medulla, pons, and the nerves issuing from these parts.

Symptoms.—In tumor the symptoms preceding the development of bulbar manifestations are the general signs of brain tumor, such as headache, vertigo, and vomiting. Only when the mass becomes of sufficient size to interfere with the functions of the medulla do bulbar symptoms appear; provided, however, that no hemorrhage has occurred therein; in that case the onset is sudden.

Aneurysm of either the basilar or vertebral arteries compressing the medulla has usually been preceded by a series of symptoms common to other forms of cerebral arteriosclerosis. In this form we are often able to elicit a history of occipital pain and impairment of the head movements. The bulbar syndrome may appear suddenly or come on gradually. There are frequent seizures of anarthria, paralysis of deglutition, dyspnea, accelerated pulse, cardiac irregularity, and occasionally rises in temperature. During the interval between attacks, symptoms may remain to indicate that pathological processes are still at work in the region of the pons-medulla. Among these may be mentioned facial twitchings, paralysis of the facial, trigeminus, acousticus, spinal accessory, and vagus nerves. According to Oppenheim, the alternating and variable character of these paralyses is distinctive of the condition. The paralysis may be of either the spastic or the atrophic type; of the hemiplegic or the paraplegic variety. Oppenheim also speaks of Gerhardt's sign as being of diagnostic import. It consists in the presence of a vascular murmur on the back of the head, which may be heard by the examiner. Another symptom is mentioned as characteristic for aneurysm of the basilar artery, which may be elicited as follows: While the patient is in the recumbent posture, breathing normally, throw his head forward; instantly there is a tendency to the stoppage of respiration in expiration; when the head is thrown backward, normal respiration is again resumed.

Prognosis.—The outlook in compression of the medulla by either tumor or aneurysm is exceedingly grave. It is hardly necessary to state that a tumor in this region is irremovable. As for aneurysm, this, too, eventually terminates fatally. While it is possible for a patient with an aneurysm at the base of the brain to live for months and even years, he is, as John Hunter expressed it, "at the mercy of every rascal who chooses to take his life." After some little excitement, or without any apparent cause, the aneurysm ruptures and instant death is the result. The only ray of hope is seen in a possible etiology of syphilis, when antispecific treatment may yield results.

Treatment

This does not differ from that of tumor of the brain situated elsewhere, except that surgery is out of consideration in pontine tumor. However, should symptoms result from large or small cerebellopontile angle tumors, surgery is not only to be tried as a last resort, but should be instituted as soon as a diagnosis has been made.

In the aneurysmal cases the treatment is identical with that of cerebral arteriosclerosis, acute bulbar paralysis, and cerebral thrombosis. I am of the opinion that moderate doses of sodium iodid have a beneficial palliative effect upon aneurysm. Great triumphs may occasionally be achieved in the syphilitic cases. Of course, there can be no limit to the amount of antisyphilitic medication which such a patient should receive. Its administration means the saving of life in a case without any other hope of recovery.

4. PSEUDOBULBAR PARALYSIS AND CEREBROBULBAR GLOSSOPHARYNGOLABIAL PARALYSIS

Introduction.—In order to understand this condition it is well to recall a few anatomicophysiological data. Following the neurone theory, each motor neurone consists of at least two kinds—an upper and a lower neurone. The nuclei contained in the medulla constitute the beginning of the lower neurone; their upper representation being situated just above and originating in the cortical cells. Similar to upper motor neurone lesions in the skeletal muscles, there is an upper motor neurone affection of the medulla, capable of producing paralysis of its functions. This disease we shall now discuss—its peripheral counterpart, progressive bulbar paralysis, having been already considered.

By pseudobulbar paralysis we mean that the central or upper representation of the medulla has been interfered with, the necessary messages no longer reach the peripheral structures, and the result is a paralysis not unlike that of the genuine bulbar variety. As long as stimuli from above can pass down, even though it be through one limb of the arc, there is no appreciable derangement of function. But no sooner has anything occurred to completely isolate the medulla from its higher centers, as, for instance, a second "stroke," than the bulbar paralysis becomes complete.

Pathology.—The pathological conditions underlying the production of pseudobulbar palsy are usually vascular changes causing either hemorrhage, thrombosis, or embolism. Further, it matters little which portion of the upper motor neurone has become implicated; the essential requisite is that the lesion must have occurred before the central fibers have arborized around the nuclei of the medulla. It is also necessary that both sides shall have become affected, either simultaneously or in succession. Oppenheim states that in many cases additional foci of inflammation or softening are found in the pons.

Etiology.—Everything which stands in a causative relation to the production of hemorrhage, thrombosis, and embolism may be considered a cause of this disease. The chief factors are: syphilis, arteriosclerosis, heart disease; insular sclerosis and multiple cysts may rarely produce the syndrome of pseudobulbar palsy.

Infantile pseudobulbar palsy yields identical symptoms. Oppenheim has described this condition in children in connection with cerebral diplegia. The infantile variety is due to an arrest of development or malformation of the lower parts of the central convolutions.

Symptoms.—In the majority of cases there is a history of a first attack of hemiplegia with perhaps some slight disturbance of phonation or deglutition. Soon a second apoplectiform seizure takes place and a complete or incomplete case of bulbar palsy becomes established. The symptoms are like those of the fully developed progressive bulbar paralysis. A patient so affected presents an expressionless, even stupid face, particularly in its lower half; an open mouth, from which saliva dribbles more or less constantly; paresis of the muscles of the cheeks, lips, tongue, palate, mastication, and of the vocal cords. In addition there are nasal intonation to the voice, dysarthria with or without aphonia, dysphagia, or even complete inability to swallow; toward the last respiratory difficulties with attacks of dyspnea may become frequent. There are a number of symptoms characteristic of the pseudobulbar form, thus distinguishing it from the chronic progressive variety. Among these must be emphasized the absence of atrophy in the paretic muscles and the preservation of reflexes and of the normal electrical reactions. Added to these there may be unilateral or bilateral hemiplegia, occasional involvement of the optic nerves—mild neuritis or optic atrophy—occasioned by the numerous arteriosclerotic foci scattered throughout the visual tracts. In a number of cases bladder and rectal disturbances have been noted. Czylharz-Marburg's researches of the sphincters, having a bilateral representation in the central ganglia, may throw some light on this symptom.

It is interesting to observe that paralysis of the muscles is not complete; movements which can no more be executed voluntarily may still be set in action by emotional causes. Speech may be impossible, but involuntary emotional responses may still be preserved. Another remarkable symptom is the modification in the acts of mimicry—the patient may have explosive outbreaks of spells of crying and laughter.

Mental symptoms are almost always present in pseudobulbar paralysis. There are usually marked impairment of memory, apathy, confusion, and quite often there is a degree of dementia present.

Differential Diagnosis.—This condition must be differentiated from genuine bulbar paralysis, acute bulbar paralysis, and the bulbar form of myasthenia gravis. The differential diagnosis has been sufficiently discussed in the previous paragraphs, and I do not think there will be any difficulty when one has mastered the few essential points.

Treatment

Antispecific medication may be given a trial even in cases that are frankly non-syphilitic in character. Even in these cases some improvement has been seen from the administration of the iodids in moderate doses, given over extended periods of time.

The symptomatic treatment is similar to that of the other varieties of bulbar paralysis.

5. MYASTHENIA GRAVIS

(*Asthenic Bulbar Paralysis*)

Introduction.—This is a disease in which excessive *exhaustion* is the prominent symptom. After a period of rest partial recovery of power often occurs, but the affected muscles are, as a rule, incapable of doing sustained work.

Etiology.—The sexes are about equally divided, and the disease appears during the first half of life. The exact etiology is still a matter of speculation.

Pathology.—Nothing definite is known even with regard to the etiological pathology of this interesting condition. With reference to the central nervous system, nothing notable has been found beyond some indefinite and inconstant changes in the cranial nerve nuclei. The view is now generally held that the essential pathology is in the muscles themselves. Weigert and others have found an exudation of lymphoid cells in the muscle substance, and this is consequently thought to be the cause of the disease. Another frequent finding is an enlarged thymus gland, which may be the seat of lymphosarcoma. No connection has been established between thymus involvement and the exudation of lymphoid cells into muscles. Most writers favor the view that the condition is caused by toxins, the origin and composition of which are still to be discovered.

Symptoms.—Weakness in the musculature is the leading complaint. As not all parts are equally affected, the symptoms will vary with the number and function of muscles involved. Variations in the intensity of symptoms are quite common, and alternating exacerbations and remissions are almost the rule. Among the most serious forms of this disease are the ocular and bulbar varieties. Of the *eye muscles,* the levator is most prone to become affected; the resulting symptom then is ptosis. The patient is unable to keep his eyes open for more than a few seconds; there is a tendency for the lids to droop and for the eyes to require frequent rests. After a short interval the patient again uses his eyes, but fatigue soon returns. In order to see at all the patient is compelled to throw his head back and even to hold up the lids with his fingers. The compensatory action of the occipitofrontalis, seen in the wrinkling of the brow—a usual feature of ptosis from other causes—is generally absent because of the rapid exhaustibility of this muscle. Transient ocular palsies of various other kinds are also observed; likewise nystagmus.

Bulbar symptoms appear chiefly in connection with the muscles of the mouth, tongue, and palate. Weakness of the orbicularis oris makes it difficult for the patient to blow or whistle, and even speech may become affected. During the states of exhaustion the tongue cannot be protruded or forcibly moved from side to side. The paralyzed soft palate no longer shuts off the nasal from the oral cavity, and a nasal voice is produced in consequence. There may also be difficulty in swallowing, with regurgitation of fluids through the nose. All the muscles seem to tire after the slightest exertion; even the masseters become exhausted during a meal, preventing its completion. The facial muscles may likewise suffer, permitting the appearance of a drooping lip. Gowers mentions as characteristic for this condition the so-called "nasal smile," in which the movement at the corners of the mouth is deficient, the furrow of the smile being sometimes entirely above the upper lip.

The limbs are frequently affected. The patient then becomes easily fatigued; walking is tiresome and manual labor impossible. Any occupation requiring the use of the hands is out of the question. The neck muscles may suffer and allow the head to fall forward on the chest. When the muscles of respiration participate, there may occur attacks of dyspnea.

An objective symptom of great importance is the so-called myasthenic reaction. This consists in a great exhaustibility of the voluntary muscles by the faradic current. When the muscles are stimulated, at first the contractions may be fairly good; after a short time, perhaps within a few minutes, the muscles become less and less responsive, until the contractions cease altogether. After resting a little the muscles again respond to electrical stimulation. It is noteworthy that, though an electrical response may not be obtained, the muscles still obey the will. The myasthenic reaction, when present, is a very valuable diagnostic sign. It varies in completeness, however, from time to time, and is not found in all muscles.

There is nothing pathognomonic about the reflexes; they may be present, absent, or reduced. When present, the knee reflexes can be easily exhausted after a few tappings. There is seldom wasting of individual muscles, though the patient may appear generally emaciated. Of sensory symptoms paresthesia may be complained of, but objective sensory disturbances cannot be elicited.

Differential Diagnosis.—The cases must be discussed as ocular, bulbar, and spinal types, according to the parts in which the first symptoms appear. When *eye* symptoms are prominent, conditions in which ptosis and partial ophthalmoplegia are symptoms must be differentiated. Tabes with ocular palsies may occasionally be mistaken for myasthenia of this type. A search for other symptoms of the disease, such as Argyll-Robertson pupil, lancinating pains, the loss of knee reflexes, and the presence of ataxia, should decide the diagnosis. Migraine with transient ocular palsies may be differentiated by the palsy always having a definite relation to the head pain. Ptosis may be a symptom of brain tumor, syphilis, or hysteria. In the latter disease the ptosis is spasmodic, and not paralytic, as in myasthenia. For tumor and syphilis there will be a multitude of other signs to clear up the differential diagnosis.

From the *bulbar* variety progressive bulbar paralysis and post-diphtheritic palsy must occasionally be differentiated. In the chronic organic bulbar disease the course is progressive, there is distinct wasting of muscles, there is no remission in symptoms, and the myasthenic electrical reaction is absent. Post-diphtheritic palsy should be differentiated by the history and course of the disease, as well as by the electrical examination.

The *spinal* type of myasthenia has frequently been mistaken for neurasthenia. By attention to details, and the golden rule never to diagnose the latter disease until every other condition has been excluded, mistakes can be avoided. Under certain circumstances progressive muscular dystrophy can be mistaken for myasthenia gravis. In both the patient tires easily and is generally weak. Of course, the localized atrophies found in the dystrophic condition would be of great assistance if always present. Early in its course, however, atrophies may not have declared themselves as yet. In that case the careful electric examination of muscles will usually settle the diagnosis.

Prognosis.—The prospects for recovery are not favorable in the majority of cases. Remissions and intermissions of long duration are the rule. In a small number of cases symptoms have permanently disappeared. Unfortunately the tendency even in those who have had remissions is toward greater exhaustibility. The patient usually dies during an attack of respiratory or cardiac failure, or succumbs during a choking spell while eating.

Treatment

In the treatment of a case of myasthenia gravis great patience is required. The ordinary method of applying electricity by means of irritating galvanic and faradic currents, as advised in chronic progressive bulbar paralysis, is contraindicated in myasthenic bulbar palsy. Such measures endanger the patient's life. No objection can be had to the application of central galvanization in very small quantities of current, which by some is claimed to be followed by great benefit.

The most important point in the treatment is the husbanding of the patient's strength, avoiding all muscular exertion. In a severe attack he must not leave the bed, nor must he be permitted to speak. As such patients have difficulty in masticating their food, and in swallowing, they do not eat sufficiently. It will be our endeavor, then, to give them concentrated, nitrogenous, and even partly digested foods, so as to keep up their nutrition to the highest point. During meals frequent pauses should be interposed, in order to rest the exhausted muscles; but patients must be constantly encouraged to conclude their meals.

The medicinal treatment consists of tonics, of which strychnia is the most popular one. There are those, however, who condemn this remedy. Oppenheim advises the use of hypophyseal extract and ovarian tablets; also the double salt of spermin sodium chlorid, hypodermically, in doses of 1 c. c. of a two per cent. solution, daily, or every other day.

6. OPHTHALMOPLEGIA

Introduction.—Paralysis of ocular muscles is encountered in a number of conditions and is a symptom in many diseases. The eye syndrome may assume such significance that it almost becomes an independent symptom-complex. For the sake of brevity we shall only discuss the acute and chronic varieties.

ACUTE OPHTHALMOPLEGIA

This variety is probably always caused by either intoxication or infection. The cases of hemorrhagic polioencephalitis superior of Wernicke seem to be part of this group. In some instances it is difficult to classify the cases in reference to whether they are toxic or encephalitic in character. This is particularly true when they result from the ingestion of poisonous meats, fish, sausages, or are caused by carbon dioxid poisoning. Acute ophthalmoplegia may also be caused by hemorrhage into the ventricles, exciting an inflammation in the nuclear region. Traumata of all kinds, especially the late forms of apoplexy, are etiologic factors in their production. The inflammatory forms of acute ophthalmoplegia are probably caused by acute infectious diseases, such as influenza. Among the several other causes may be mentioned syphilis, tuberculosis, alcoholic intoxication, ptomains from decayed fish and meat. Lead and other inorganic poisons may likewise have a selective effect upon the ocular nuclei.

Symptoms.—When the result of hemorrhage, there will probably be a sudden onset, with vomiting and convulsions, followed by the appearance of paralysis in one or more ocular muscles. In cases due to acute hemorrhagic polioencephalitis, prodromal signs, such as general malaise, headache, vertigo, and vomiting, may precede the development of the disease proper. The temperature is variable; it may be high or low, or remain within normal limits. Shortly after the development of the first symptoms, paralysis of the ocular muscles appears; the muscles usually escaping are the levator palpebræ superioris and the sphincter of the iris. Headache and vertigo may continue and may even become worse. If the case proceeds toward a fatal termination, stupor and coma are added. Acute ophthalmoplegia is usually bilateral and may occur in association with poliomyelitis or with the paralysis of face, tongue, and palate, due to the nuclear involvement of acute bulbar palsy.

Differential Diagnosis.—From neuritis of the ocular nerves this affection is distinguished by the presence of convulsions, muscular twitchings, headache, stupor, or coma. It must be remembered that this syndrome may actually be due to neuritis and that the diagnosis between the two may be impossible; the existence of neuritis in another part of the body makes this diagnosis the more plausible. A differentiation may have to be made between the acute variety of ophthalmoplegia and the ocular form of myasthenia gravis. The difficulty will be overcome when one thinks of the myasthenic reaction, the fatigue of muscles and nerves, capable of being temporarily, at least, removed by prolonged rest.

Prognosis.—The disease is often fatal; the most favorable cases being those following infectious diseases and the neuritic types. In those who recover paralysis may remain permanent.

Treatment

This is identical with that given previously for the treatment of acute hemorrhagic encephalitis. Ice to the head and derivative remedies to the lower extremities, including the administration of calomel and other cathartics, should not be omitted. To allay the extreme restlessness, bromids and small doses of morphin are indicated. In debilitated cases stimulants should not be withheld. Lumbar puncture is of distinct benefit when there is increased intracranial pressure. During convalescence invigorating tonics are indicated to restore the paralyzed muscles.

CHRONIC OPHTHALMOPLEGIA

Etiology.—This variety may constitute the end result of a case of acute ophthalmoplegia, in which degenerative changes have taken place. Further, chronic degeneration of the nuclei may be due to syphilis, diabetes, diphtheria, or it may be an associated symptom of tabes, paresis, multiple sclerosis, progressive muscular atrophy, or chronic bulbar palsy. Chronic ophthalmoplegia may appear as a congenital affection or as an hereditary disease.

Pathology.—The lesion is a chronic degenerative or inflammatory process found in the floor of the third ventricle and the aqueduct of Sylvius, which causes atrophy of the ganglion cells of the nuclei there situated. The pathological process is similar to that occurring in progressive spinal muscular atrophy and chronic poliomyelitis.

Symptoms.—The disease is characterized by a gradual and progressive development of paralysis involving one or more ocular muscles. One of the earliest symptoms is diplopia, which may be transient at first. Somewhat later other muscles become affected, often irregularly and without reference to function. Ptosis may be absent or incomplete. The paralysis may cease to progress and may remain more or less stationary. The more probable course is for the pathological process to implicate all the eye muscles. The disease may be unilateral, or it may affect both eyes. In some cases only the external muscles are involved; in others only the internal muscles.

Differential Diagnosis.—This can only be made from the etiology and from coexisting symptoms. For the nuclear forms no definite diagnostic criteria can be given, excepting, perhaps, the common experience that the inner eye muscles are usually spared and that ptosis is rarely present or well defined. Symptoms are almost always bilateral. Not infrequently the chronic nuclear disease follows other cerebral or spinal diseases, pointing to degenerative conditions of cortex or cord, as in general paresis, tabes, bulbar myelitis, or poliomyelitis.

Prognosis.—The chronic cases often have a protracted course and may last for years. They may, however, become completely arrested in their progress.

Treatment

This will largely depend upon the cause. If alcohol is an etiologic factor, it must be withdrawn and abstinence substituted therefor. In addition all causes must be avoided having a tendency to produce congestion of the brain. Syphilis being a common antecedent in these cases, it is well to institute a rigid course of antispecific treatment, consisting of both mercury and iodids, as described under Syphilis of the Brain. In the presence of a local cause, such as tumor, the outlook is unfavorable. If chronic ophthalmoplegia is part of a degenerative nervous disease, such as tabes, general paresis, or bulbar paralysis, the prospects are still worse. In all cases iodids in good-sized doses may be tried, as there is nothing better at our disposal, and we are never certain that even in an apparently non-specific lesion there is not some specific etiology. Treatment by electricity has its advocates; likewise hydrotherapy. Any of these measures may benefit the patient's general health, including the nervous lesion.

It is my opinion that a systematic course of strychnia in gradually increasing doses, beginning with a thirtieth of a grain, and increased up to one-twentieth, three times daily, should be tried for several months.

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CHAPTER XVII

DISEASES OF THE CEREBELLUM

JULIUS GRINKER

Various diseases of the cerebellum have already received brief mention in connection with corresponding cerebral diseases. Hemorrhage, softening, inflammation, abscess, and tumor of the cerebellum require no separate description in this place. Hereditary cerebellar ataxia and atrophy and sclerosis of the cerebellum must still be discussed.

1. ATROPHY AND SCLEROSIS OF THE CEREBELLUM

Congenital smallness of the cerebellum may be due to absence of lobules, or of a whole hemisphere; the entire cerebellum has been found in a rudimentary state.

There are also *acquired* forms, which result in shriveling, induration, and atrophy of the whole cerebellum or of some of its parts. Stated differently, there are both developmental and focal diseases of the cerebellum, which may occur in fetal and in extrauterine life. Some of the pathological changes are entirely vascular—hemorrhage, softening, inflammation; others are meningeal in character.

In addition there is a *cerebellar* type of *infantile palsy,* with lesions in the cerebellum, instead of in the brain. In some of the cases the disease comes on acutely as a severe brain affection, but leaves behind a permanent paralysis. The children are unable to walk years after the onset of the disease, and can be observed crawling on all fours. The disordered functions are probably of the cerebellar coördinating mechanism. An acquired case of this kind has been studied by Oppenheim and Arndt. They found sclerosis and atrophy of the corpus dentatum in the post mortem examination.

The phenomena observed in different instances of cerebellar atrophy were not always the same. In the majority of the cases one or more of the following symptoms were observed: defective mental development, cerebellar gait, vertigo, dysarthria, scanning speech, intention tremor, and ataxia of speech muscles. Several observers have mentioned the existence of exaggerated reflexes. Epileptic attacks, paralysis of ocular muscles, as well as abnormal position of eye muscles have been attributed to disease of the cerebellum. According to Oppenheim, the cerebellar ataxia, vertigo, and perhaps also speech disturbances might have been caused by the cerebellar disease. All the other symptoms were probably produced by hydrocephalus or cerebral involvement. Mental signs, found in several instances, may easily be explained by the hydrocephalus which was also present.

Atrophy of the cerebellum has been detected in several cases which presented symptoms similar to Friedreich's disease. Nonne, for instance, described a family disease, in which he found post mortem an abnormal smallness of the entire central nervous system. The disease had developed in three brothers either at puberty or later, and ran a chronic course in all. The symptoms were: loud and explosive speech, nystagmus, imbecility, paralysis of ocular muscles, simple optic atrophy, incoördination, and increased reflexes. It is not likely that all of these symptoms were due to abnormal smallness of the cerebellum.

Treatment

Vascular accidents of the cerebellum require treatment similar to that of the cerebrum, already described under another heading. Likewise the treatment of cerebellar inflammations differs in no essential from that of the cerebral forms. It goes without saying that developmental errors are not amenable to treatment.

2. HEREDITARY CEREBELLAR ATAXIA

(*Hérédo-ataxie Cérébélleuse*)

Introduction.—Marie first described this disease under the name hérédo-ataxie cérébélleuse and pointed out the differences between it and the hitherto well known disease, Friedreich's ataxia.

Pathology.—The cerebellum is smaller than normal, owing to an arrest of development. The medulla and spinal cord have also been found in an atrophic condition. In Marie's first case there was, besides, sclerosis of Goll's tracts, Gower's columns, and of the direct cerebellar tracts. The middle cerebellar peduncle was markedly reduced in size. In one or two instances the gray matter of the cerebellum was seen to be pathologically altered. Rarely is the cerebellum affected without other portions of the nervous system.

Etiology.—There is usually a neurotic family history. Among other things, alcoholism, tuberculosis, and consanguinity in the parents, as well as syphilis, have been made responsible for this affection. Infectious diseases and traumatism are said to play an important rôle in its causation. Members of the same family frequently develop the disease at about the same age; females are more often affected than males.

Symptoms.—Incoördination is the characteristic and striking symptom. In the majority of cases the lower extremities are affected before the upper. There are seen the reeling gait, with asynergy, asthenia, ocular disorders, and exaggerated reflexes; in fact, the usual symptoms of cerebellar disease are observed in this affection. Its onset is rather insidious. For some time previous to the development of equilibratory disturbances the patient may have complained of neurasthenoid symptoms. Soon headache, back pains and a general feeling of lassitude make their appearance. In rapid succession are then noticed typical cerebellar ataxia and peculiar speech disturbances. The patient's voice is so changed that speech is either explosive, monotonous, guttural or unintelligible. As the disease progresses, the upper extremities also become incoördinate. The arms and hands are uncertain in all movements, and tremor is developed upon activity; eventually writing and sewing become impossible. Not only in the physical but also in the mental sphere are disturbances present. Memory is considerably impaired, the patient becomes apathetic, irritable, and indifferent to his environment.

Prognosis.—The tendency is for the disease to become progressively worse, though long remissions have been observed. During the last stages the patient remains helpless in bed and usually dies of some intercurrent complication.

Differential Diagnosis.—The history and onset, as well as the gradual development of symptoms, and the hereditary and family characteristics enable the differentiation to be made from tumor, hemorrhage, or abscess of the cerebellum. Some difficulty may be experienced in distinguishing this disease from Friedreich's ataxia. The most important differential sign is in the deep reflexes: their presence in Marie's disease and their absence in Friedreich's ataxia.

Treatment

From the therapeutic viewpoint an apology is due the reader; there is no treatment. Following the vague assumption that some cases may be due to a syphilitic ancestry, antispecific treatment may be tried in the usual manner.

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CHAPTER XVIII

NEUROSES

NEURASTHENIC AND PSYCHASTHENIC STATES, INCLUDING THE PHOBIAS

General Treatment

Lewellys F. Barker and Charles M. Byrnes

Introductory.—That great difficulties are encountered in the classification of functional nervous diseases, every physician who has studied nervous patients knows. In the present section, it is our purpose to consider the treatment of the so-called neurasthenic and psychasthenic states (exclusive of the hysterical states and the psychoses).

In the neurasthenic, and especially in the psychasthenic states, the symptomatology is predominantly mental. The states are spoken of as "nervous affections" or "neuroses," a terminology which is justifiable since the patients and the patients' friends usually have a horror of mental disease. Physicians, however, should never permit this fear on the part of the laity to distort their own view of such cases; medical men should recognize that the symptoms presented by these patients are largely abnormal sensations, feelings, and psychic reactions. Neurasthenic and psychasthenic states belong in the broad borderland between mental health and outspoken mental disease (insanity). We must regard them as mild forms of mental disorder, for one sees, in practice, every transition from such states to the more serious mental disorders which we designate as psychoses. But mental disorders—mild and severe—are to be regarded as cerebral diseases; a disturbed mentality means abnormality of cerebral function. There may be no such thing as a *purely* psychic disorder, for in scientific medicine, most men work on the assumption that psychic manifestations and cerebral processes go parallel with one another. Neurasthenic and psychasthenic states are often spoken of as "functional" nervous diseases, sharply separable from the so-called "organic" nervous diseases. This is a purely arbitrary division. In both sets of diseases there may be material changes in the nerve cells. The changes in the cells, if such exist, in the so-called functional nervous disorders are so slight—possibly molecular—that they are not demonstrable by the means at present at our disposal. Too much stress should not, however, be laid upon this view; indeed, as Meyer especially has emphasized, there is some advantage to be gained in studying the mental facts as such, without considering the possibility of "lesions" or "diseases" underlying them. Different workers may well approach the problems of abnormal mentality in different ways and, in the end, all contribute to their solution.

Another point to be kept ever in mind is this: the neurasthenic and the psychasthenic states are not to be regarded as "diseases" *per se* so much as nervous and mental syndromes—abnormal mental attitudes and mental reactions—producible by a variety of causes. Every physician knows how frequently a neurasthenic or a psychasthenic state presents itself at the onset of some organic disease in some other part of the body; the primary organic disease may affect, perhaps, an organ far removed from the nervous system, or from the parts in which symptoms are first complained of. Examples of such symptomatic neurasthenic or psychasthenic states may be met with at the beginning of a pulmonary tuberculosis, in association with a chronic inflammation of the paranasal sinuses, in the larvate forms of exophthalmic goiter, in chronic arthritis of the spine, in abnormalities of the eye (refraction errors; muscular insufficiencies), in the early stages of brain tumor, in tabes and general paresis, in cerebral lues, in pelvic inflammatory disease, in anemia, in visceroptosis, at the onset of some of the psychoses (dementia precox; manic depressive insanity), in various intoxications (abnormalities of internal secretion, gout, diabetes, chronic constipation, drug habits, alcoholism, tabagism), in atherosclerosis, etc. For this reason it is desirable that every patient complaining of neurasthenic or psychasthenic symptoms should be subjected to a most careful general clinical study, including a thorough examination of all the organs of the body, as well as of those of the nervous system proper. A neurologist who confined his examinations to the testing of the nervous and mental functions only, would sometimes overlook the existence of one of the diseases mentioned. The importance of a thorough training in inner medicine for all neurologists, and of a good training in neurology for all who work in inner medicine, is thus emphasized. He who undertakes the treatment of neurotic patients should be skilled in all the modern refinements of diagnosis and should exhaust them in the study of his case, or have some skilled physician do so for him, before beginning his therapy.

In the *neurasthenic states*, the most constant symptom is fatigability, often accompanied by headache, or a sense of pressure in the head, pain in the back, and insomnia. The patients are often depressed mentally, and tend to focus their attention upon slight disturbances in the digestive apparatus, in the circulatory apparatus, or in the genitourinary apparatus.

In the *psychasthenic states,* the patients suffer from sensations of incompleteness, from disturbance of the feelings of reality, and from other symptoms referable to "lowering of the psychologic tension." Among the characteristic phenomena met with in psychasthenia, so carefully studied by P. Janet, may be included (1) obsessions, (2) pseudohallucinations, (3) abnormal impulsions, (4) mental manias, (5) ruminations, (6) tics, (7) forced agitations, (8) phobias, (9) deliria of contact, (10) anxiety conditions, (11) sense of strangeness and unreality, and (12) phenomena of depersonalization.

Phobias are met with clinically in immense variety. The classification of phobias proposed by Janet is very good. He divides these pathological fears into four great groups: (1) the algias, and bodily fears, (2) the fears of objects (*délire du contact*), (3) the fears of situations (agoraphobia), and (4) the fears of ideas.

Among the *phobias of the body* are included (a) the *algias* in different parts of the body (chest, skin, head, feet, hands, limbs, genitals, bladder, anus, etc.), and (b) the *phobias of bodily function* (movements, writing, walking, eating, swallowing, digesting, defecating, breathing, speaking, smell, hearing, sight, etc.).

Among the *phobias of objects* are included the fears of dangerous objects, of dirt, of people, of animals, of professional instruments, etc.

Among the *fears of situation* are included (a) fears of physical situations (agoraphobia, altrophobia, claustrophobia), and (b) fears of *social situations* (fear of blushing, fear of looking peculiar or of acting strangely, fear of servants, fear of marriage, etc.).

Among the *fears of ideas* may be included (a) fears of religious ideas, (b) fears of moral ideas, (c) fear of death, (d) fear of insanity, (e) fear of disease, etc.

With some patients instead of these systematized, emotional, compulsory agitations, the fear takes a diffuse form, either that of *physical anxiety* (digestive, circulatory, or respiratory), or of *general mental anxiety.*

When reading in the literature of the subject, some attention must be paid to the different ways in which terms are used by different neurologists. Thus, some apply the term *neurosis* or *psychoneurosis* to any one of the so-called functional nervous disorders. Aschaffenburg and Binswanger (5), in Germany, and Janet, Déjerine, and Bouveret, in France, emphasize especially the psychic side of these cases. But while Aschaffenburg would include hysteria, psychasthenia, and neurasthenia all under the general term, psychasthenic states, Janet sharply distinguishes hysteria from psychasthenia. In America, Beard emphasized the independence of neurasthenia as a disease entity, while the majority of writers (especially Dana, 14; Bartholow and Browning, 9) have stressed its symptomatic nature.

Freud (19), of Vienna, excludes the symptomatic neurotic states like those occurring in Basedow's disease, in tetany, and in chorea, from the neuroses proper. He divides the neuroses, thus restricted, into two great groups: (1) the "actual neuroses," and (2) the "psychoneuroses." They are all, in his opinion, of sexual origin. The "actual neuroses" include (a) "neurasthenia proper," and (b) the "anxiety neuroses"; the psychoneuroses include (a) hysteria, and (b) the so-called "compulsory neuroses." Freud's classification is based upon his view that in the "actual neuroses" some abnormal sexual activity is going on at the time, while the psychoneuroses, he thinks, are due, not to abnormal sexuality at the time the symptoms occur, but to abnormal sexuality in early childhood. Freud further states that the symptoms in the "actual neuroses" appear to be toxic in nature, while in the "psychoneuroses" they are "psychogenic" in origin, depending upon the activity of unconscious (suppressed) idea-complexes of sexual-erotic content arising from the sexual needs of unsatisfied individuals, the symptoms representing a sort of "substitute satisfaction." Freud admits also the existence of *mixed cases* in which a compulsory neurosis is combined with neurasthenia or an anxiety neurosis with hysteria; in these mixed cases he assumes a combined etiology. Freud believes that, in hysteria, the symptoms are due to the permanent action of psychic complexes, the emotional accompaniments of which have become separated from the patient's consciousness and lead to abnormal innervations of the body (phenomena of "conversion"). In the compulsory neuroses (psychasthenic states), on the other hand, though the emotion has become separated from its original sexual idea, it is not "converted" into abnormal bodily innervation, but attaches itself, he believes, to some other idea, in itself indifferent, thus giving rise to an obsession or a phobia (phenomena of "substitution"). According to this view, both hysteria and the compulsory neuroses are instances of unsuccessful efforts at "defense" (*Abwehr*), of unlucky attempts to drive painful ideas and emotions from consciousness (*Verdrängung*).

In considering the treatment of the neurasthenic and psychasthenic states it will be convenient to consider, first, prophylaxis and the general somatic treatment, and, afterward, to take up the more special psychotherapeutic treatment.

Prophylaxis

While the prevention of disease is the physician's highest aim, most preventive measures require, to be effective, not only an enlightened medical opinion, but also an educated and coöperative public sentiment. This is especially true in regard to abnormal psychic states. Before much can be expected from prophylactic measures, the physician must learn how to detect the earliest manifestation of neurotic conditions, and the laity should have, at least, some understanding of the normal processes of psychical development and of the conditions which tend to abnormality.

Race.—Heredity undoubtedly furnishes the foundation for many of the neuroses. Thus, it is well known that certain races show marked susceptibility. The Hebrews, Slavs, Poles, Russians, and Americans are particularly liable. This innate tendency reveals itself when these races are exposed to hardship and strain. It is especially noticeable among members of the immigrant class who make their homes in the crowded districts of our larger cities. Many of them arrive when quite young, almost without money, and are forced to accept any employment that will insure a bare livelihood. Not a few of them have broken home ties on account of intolerable social and domestic conditions which have already left telling marks upon their unstable constitutions. Conditions in the New World are not what were expected. In time, the faulty social, economic, and hygienic surroundings overcome the small physical and mental reserve, and "neurasthenia" is established. It is now too late for prophylaxis and treatment is difficult. If the existence of conditions like these were more fully recognized and appreciated, and a proper study made of the surroundings of such people, much could be done by society to protect from too great environmental strain those who have inherited bad nervous systems; as it is, despite the beginnings made by workers for social betterment, the victims too often fail to attract attention until forced by their ailments or incapacity to apply for aid to a neurological dispensary or a Social Service Committee.

Eugenics.—But it is not only in the humbler walks of life that heredity is important as an etiological factor. No race, class, or condition is immune from abnormal psychic states; and if we are to apply what we know regarding hereditary transmission of a neuropathic tendency, it is clear that prophylaxis should really begin before birth. The thoughtful physician will deprecate marriage or, at any rate, parenthood, among those with decided hereditary neuropathic and psychopathic tendencies. Bouveret (7) and Godlewski (23) have expressed strong opinions in discussing this question of eugenics. The former states that marriage should be prohibited in the hereditary type of neurasthenia. It may, he thinks, be permitted in the acquired form, however, provided the patient has improved and there is no nervousness in the family into which he or she marries. Godlewski thinks that marriage between two persons, both of whom are neurotic, should be regarded with disfavor. Should only one be neurotic, the children are not so likely to suffer. In his opinion, rheumatism is so frequently found to be an etiological factor in the neuroses that marriage among rheumatics should be discountenanced.

In some states in America, laws have already been passed, providing for the denial of the privilege of parenthood to the "manifestly unfit" (habitual criminals; epileptics; the feeble-minded; the insane; chronic alcoholics). In Sweden, an attempt is being made especially to provide for parents of good stock among the peasant classes, with the hope of improving the race.

Infancy.—Where there is a history of neurotic tendencies in one or both parents, it is wise to institute prophylactic measures early. A careful régime in infancy, important for all children, no matter what their parentage, becomes doubly important where there is the probability of neuropathic taint. We must insist upon regular feeding, see that nourishment of proper quality and in right quantity is given, clothe the child suitably, and provide for sufficient sleep, and plenty of fresh air. Much good, in this direction, will doubtless result from the campaign of education now being carried on by the American Society for the Prevention of Infant Mortality. Much harm is done by overindulgent, solicitous mothers who constantly fondle, walk with, nurse, or rock a child every time it utters an unfamiliar sound or shows the slightest restlessness. Mothers and nursemaids should be taught to begin early to instill regularity of habits and to require obedience. A child very easily acquires habits—either good or bad; whether they be good or bad will depend almost wholly, when the brain is normal, on the parents and nurse.

Childhood.—In general, a similar régime should be continued throughout later childhood, with adaptations, of course, to the conditions of life during this period. It is during childhood that the individual is most impressionable and plastic; at this age, correct habits of living are easily established, but, unfortunately, defective modes of living are just as readily learned, and these may, later, be serious obstacles to any effort directed toward nervous and mental hygiene. Every physician is familiar with the overindulged, self-centered, domineering child, who really directs the affairs of the household and must have its own way in all affairs of life, the child which, on the slightest opposition to its will, may exhibit any one of a variety of moods and passions, from sulkiness to the most violent outbursts of temper. How often, in such a case, a naïve, misinformed, and perhaps neurotic mother, or father, will make all manner of excuses for the child, saying that it has always been delicate, that it does not feel well, or that it inherits its disposition, and cannot behave otherwise. Such parents, overflowing with shortsighted and culpable sentimentality, quote to themselves the lines:

> "Teach me to feel another's woe.
> To hide the fault I see,"

and let the child have its own way. Taking the child to task, admonishing it, they tell you, only makes things worse. It may be argued that such a child is already in the nervous class, and to a certain extent this is true, the ear marks of "nervousness" are present, but there is usually ample time even then to overcome the manifestations by the application of carefully selected prophylactic and therapeutic principles. Such children should be taught obedience, self-denial, stoicism, and responsibility. Strict attention should be paid to rest, sleep, fresh air, moderate exercise, the daily sponge-bath of cool or cold water, and to the diet and bowels. The use of coffee, tea, and stimulating drinks should be prohibited. Self-abuse should be strictly guarded against. It may be even necessary to place the child away from home in the care of some one other than an immediate member of the family, though a well-chosen governess or an intelligent nurse may accomplish much. Whoever takes charge should be kind, firm, but not harsh, truly sympathetic without indulging unduly, an abnormal craving for sympathy. If possible, she should have had some experience in dealing with nervous or ill-directed children. Excitement, competitive games, and over-vigorous exercises are particularly harmful to nervous children.

Education in school is an important feature in the lives of such children. It should not be begun before the seventh year, and great care should be taken to avoid forcing nervous children beyond their strength at school. If conditions in the family circle are decidedly unfavorable it will be wise to have the child educated away from home, say in a good boarding school, where system, regularity, obedience, and outdoor exercise form a large part of the educative process. An "only" child is in great danger of being spoiled and of becoming nervous, for children need the educative influences of companions of approximately their own age. The "only" child should in some way be thrown regularly into contact with other children.

Adolescence.—If the preceding prophylactic measures have been faithfully observed, good habits should have been sufficiently established to carry the individual safely through the adolescent period. There are certain dangers, however, attendant on the awakening of sex consciousness: the transition from childhood to manhood, or womanhood, demands careful supervision. The physical and psychical changes of this period are more or less impressive, and often make their appearance as a surprise to a wholly uninformed child. This is particularly true of girls, who, because of false modesty and their mothers' aversion to matters sexual, have never been told about the normal process of this period of life. Many children are away at boarding-school during this period, and the realization of the sexual side of life comes as a shock, especially to sensitive girls. Mysterious and vague ideas are engendered; self-inspection, masturbation, or other abnormal practices may get a start. Children approaching this period should be given, in advance, a plain, sensible explanation of the evolution of the sex instinct.

The physical, mental, and moral education of the adolescent requires special attention. In the neuropathic temperament, mental and physical work should be subject to strict regulation, and the former should occupy a secondary place. Well-chosen, systematic exercise should be urged. Rowing, swimming, tennis, running, riding, and gymnastics under a trained instructor are often beneficial. A system of disciplinary physical training like the so-called "dromotherapy" of Burlureaux, at Auteuil, in which the individual needs are carefully studied and the exercises prescribed with an intelligent regard for grade, speed, and duration can be recommended highly.

If the child is to be educated away from home, the choice of a school is of importance. If possible, a school should be selected in which a certain amount of physical exercise is compulsory; it should be located, preferably, in a smaller town, or a rural district. In France, the Academy of Medicine has demanded a certain supervision of the physical education of school children. Within the last few decades, there has developed, everywhere, greater regard for the necessity of bodily development. In England, Eton, Harrow, and many of the older institutions of learning, are located in the smaller towns, or within easy reach of the country districts. The hours of study have been shortened, and the more moderate athletic games occupy a prominent part in school life. In America, preparatory schools, State universities, and many other seats of learning form, not infrequently, nuclei about which small towns develop. In such environments, there are fewer distracting influences; teacher and taught are more intimately associated, and the chief diversion is usually some form of outdoor sport. Many young boys who have failed to learn the lessons of obedience, punctuality, and respect at home show marked improvement after a shorter or longer residence in one of our preparatory schools in which military régime is an essential feature. The problems of this time of life are well described in Stanley Hall's volume "Adolescence" (26), and the book may be read with advantage by all who are interested in the prevention of nervous disorders.

Adults.—Effective prophylaxis in the adult is a much more difficult problem.

In the hereditary types, unless rigid precautions have been employed early, the abnormal states have most likely made their appearance by this time, and we have to do with the treatment of an established neurosis, rather than with its prevention. Should, however, the individual have reached adult life safely, it becomes necessary to protect him, guided by knowledge of his personality and his ancestry, as far as possible from the abnormal or indiscreet methods of living which are most likely to undo an individual of neurotic temperament.

In the attempt to ward off the acquired neuroses of adult life, the physician enters a more favorable field for the application of preventive measures. If any primary state—visceroptosis, anemia, status lymphaticus, defective metabolism, chronic infection—prone to be associated with neurosis be detected, it should be corrected. Overwork, worry, and a sedentary life should be avoided. The over-ambitious should be held in leash, and those subject to strong emotions and passions educated to self-control. Social, or intellectual, aspirations may, for a time, have to be discouraged. Convalescence from infectious diseases should be particularly guarded; this is to be especially advised after influenza, typhoid fever, and malaria.

The engagement period is one of great strain; "premarital neurasthenia" is its expression. The marriage ceremony is, in itself, a most disconcerting affair to the self-conscious, the self-centered, the neurotic. The numerous preparations that must precede it; the anticipation, anxiety, and misgivings regarding the ceremony in public and the subsequent consummation of marriage, are very trying experiences for the neurasthenic. The physical side of the sexual relations often comes as a severe shock to a sensitive woman. Almost inconceivable as it may appear, it is undoubtedly a fact, that numbers of women enter upon marital relations wholly uninformed as to the nature and function of sex!

The neurotic woman should be carefully guarded during gestation and the puerperium. All depressing and exciting emotions should be avoided; fright, anxiety, worry, or chiding may at these times be very harmful.

Childbirth, in *primiparæ,* is always a trying experience;. much assistance may be given to an apprehensive woman by a conversation beforehand; the more usual phenomena of delivery should be fully explained; the obstetrician should see to it that proper hygienic and dietetic measures are instituted; the prospective mother should be given a reasonable assurance of a happy termination of the pregnancy. A thorough examination by a competent obstetrician, in whose opinion the patient has confidence, is usually reassuring.

The menopause, or climacteric period, is known, even to the laity, as a critical time of life. Between forty and fifty, women not infrequently complain of "nervousness" which they attribute to the "change of life." No doubt the nervous symptoms in many cases can be avoided, or greatly ameliorated, by watchful, intelligent care, and by suitable physical and mental hygiene, during this regressive stage.

Preliminary Therapeutic Reflections

Before prescribing any form of treatment for the neurasthenic and psychasthenic states, or even consenting to assume the direction and supervision of patients suffering from them, certain general factors which pertain to their therapy demand consideration.

Psychology a Valuable Adjuvant.—Neurasthenic and psychasthenic states, whether primary or secondary, congenital or acquired, are most certainly manifestations of abnormal psychic activity. What is a state of consciousness? How are ideas associated? Why do people think, feel, and act in more or less different ways under what seem to be precisely similar external conditions? The exact physical answer to these questions may never be known, but the psychic facts themselves are accessible to analysis, and a study of psychology may help us to understand, in a general way, the laws to which these fundamental processes of our mental life are subject. Every one who intends to undertake the treatment of abnormal nervous and mental states should familiarize himself with at least the elements of psychology. Individuals differ, perhaps, more in their mentality than in their external physical features—in fact, individuality itself is largely a matter of psychic potentiality. A normal heart sound, a normal pulmonary resonance, or a normal respiratory murmur are capable of demonstration; the physical facts are sufficiently constant to furnish us with a standard which we designate as normal. We may also know fairly well how one with a sound mentality will act under ordinary circumstances, but we cannot know the thoughts which are aroused in another by the objects he passes, for we can never appreciate fully the total background of experience of another. In no two patients suffering from functional nervous disorders are the psychical or the bodily conditions exactly alike; we do well on meeting them to keep one of Beard's aphorisms clearly in mind, namely: "Each case of neurasthenia is a study in itself. No two cases are alike in all details. If two cases are treated precisely alike in all details from beginning to end, it is probable that one of them is treated wrong" (3).

A Correct Diagnosis the First Essential.—From a therapeutic standpoint, the nervous symptoms may be classified as mild, or moderate, but we should never be satisfied with the mere diagnosis of the existence of a neurasthenic or psychasthenic state. The diagnostic study, on both the physical and psychical side, should be thorough-going, and should consider not only the possibility of anatomical lesions, but also, and more especially, the pathological physiology, the abnormal psychology, and the etiology. In each case, every effort should be made to discover the presence of some primary abnormal physical condition as a basis. If detected, rational treatment must be directed toward correcting it. Not infrequently a neurasthenic or psychasthenic state may be detected, or at least surmised, the moment a patient enters the consulting room. His attitude, demeanor, manner of entering the room, and method of relating his symptoms all contribute to the diagnosis. But even though the nervous phenomena may be outstanding, the dangers of "snap diagnosis" and hasty conjectures should be remembered; they too often lead to actual error; and, moreover, even could one be sure, a diagnosis announced before a thorough physical examination has been made, makes an unfavorable impression upon these victims of nervous instability. Neurasthenic and psychasthenic states, as primary conditions of the nervous system, should be our last, rather than our first, consideration. Every nervous patient should be approached with an open, unbiased mind, eager to detect some tangible cause of the symptoms, but, at the same time, conservative in its judgments regarding causal relationships.

A careful history should be taken, and a thorough physical and mental examination made. This is often impracticable at a single office visit, and may entail much work, including the making of many laboratory tests. For this reason it is advisable to have such patients, and particularly those who do not live near by, enter a general hospital for a few days, where a thorough study, including the various special examinations, may be made. Should some minor physical abnormality be detected, say an enlarged turbinate, a tender ovary, a sensitive prostate, a variable gastric acidity, great caution should be exercised in assigning to it an etiological rôle; unless we are perfectly sure of a causative relation, too emphatic a statement regarding cause should not be made to the patient. An unguarded opinion, thoughtlessly expressed, often serves merely to supplant one pathological idea by another equally as tenacious; occasionally, it is to be feared, physicians are, in such cases, led to institute useless, and even harmful, local treatments, when they would have done far better systematically to have neglected local symptoms and to have directed their attention almost wholly to general upbuilding treatment. We feel that, in this connection, a word of caution should be said, too, about operative procedures in neurotic states.

Surgery and the Neuroses.—Specialization in medicine has its drawbacks as well as its advantages, and the neurotic, especially, are not infrequently the victims of "over-specialization" and injudicious surgical interference. A narrow "specialist," with inadequate ability, training, or experience, may be tempted unwisely to anchor a "floating" kidney, to cauterize a slightly enlarged turbinate bone, to shorten the mesentery of a slightly prolapsed viscus, to cut out the colon for constipation, to dilate and curette the uterus for a menstrual disorder, to cut an eye muscle in the faulty convergence of hyperthyroidism, to apply cumbersome apparatus to a "nervous" joint affection, etc. Of course, neurologists and internists are also sometimes blameworthy in overlooking, or underestimating, local defects which are really important. Medical judgment may be sorely taxed in a given case. Even in outspoken organic disease, it is safer, sometimes, to institute general measures for a time, and to treat the patient's neurotic state until sufficiently improved later to undergo operative treatment, if necessary, without serious risk of augmenting the nervous condition. The decision of such matters requires careful and unselfish consideration on the part of the physician and surgeon. The most conscientious men—internists, surgeons, rhinologists, gynecologists, neurologists, etc.—will sometimes err. We should all of us remember that specialization tends to contract the visual fields, to lead to the old fallacy of attempting *aus einem Punkte zu kurieren*—to cure everything by treating one part!

First, let us avoid all unnecessary interference. If operation be decided upon, let it carry with it a strong conviction that it will be followed by relief. Careful post-operative care is also an essential feature in neurotic patients. James G. Mumford, in a paper read before the American Surgical Association, adduced some interesting observations upon this subject. He reviewed the histories of five hundred patients, eight years after their discharge from the surgical service of the Massachusetts General Hospital. Of this number one hundred and twenty-nine could be communicated with; he concluded from their reports that if there were more regard for post-operative care in surgical cases, and if a longer period of personal supervision were maintained, there would be fewer instances of semi-invalidism following upon sojourn in the surgical wards in our general hospitals. Patients might fare better if busy surgeons would, more often, turn over patients in convalescence from operations to their medical colleagues for supervision.

Selection of a Therapeutic Régime.—After coming to a conclusion as to the relative importance of any physical defects discovered in a "nervous" patient, and in how far we are to have recourse to general measures in combating the neurasthenic or psychasthenic state, we must ask ourselves the question: How can we best apply our therapeutic principles to the particular individual before us? The answer to this question will depend largely upon a number of circumstances: the severity of the symptoms, the sex, the state of the patient's domestic life, and the patient's financial condition. Though we may have positive ideas as to what should be done, were the patient to be put under the very best conditions, it may easily be that such conditions are more expensive than the patient can afford.

Thus, if the patient be a male, upon a small salary, and his income is the only source of revenue for a growing and dependent family, the customary hospital, sanatorium, or rest-cure treatment, with the additional expenses of a physician's fees, may be wholly unsuitable. The mere consciousness of the fact that a prolonged course of therapy in these circumstances means hardship for those dependent upon him, would only aggravate his burden, as the expense and loss of income would be a constant source of worry. In such instances, from the more elaborate methods of treatment, measures should be selected that are compatible with the patient's social and economic condition.

If the symptoms are not very severe, and especially when the features are psychasthenic rather than neurasthenic, a great deal of benefit frequently follows upon merely the thorough preliminary study of the case. This is often seen, when, after completing the examination, we tell the patient frankly, and in simple language, the nature of his ailment. A patient may be greatly relieved when, after a thorough physical examination has been made, he can be told by his physician that no abnormality outside of a functional disturbance of the nervous system has been discovered which could account for his symptoms; that, though this is true, it is realized that he is ill, and that his symptoms are not imaginary, though they may be largely psychical, and due to misrepresentation of purely normal stimuli acting upon an over-stimulated or exhausted nervous system. One must, of course, use the words "psychical" and "mental" cautiously, lest he excite fears of serious mental disease or insanity. It is especially helpful to a patient if the physician can conscientiously say that the symptoms seem to him to indicate a form of nervousness which is curable. Such a conversation, alone, in many cases accomplishes much, for it assures the patient that his suffering is understood, allays his fears, and inspires confidence and hope of recovery. When actual organic visceral disease is found to exist, and it, in the judgment of the physician, is responsible for the nervous symptoms, the facts should be explained to the patient, care being taken to excite no unnecessary alarm or apprehension. Treatment of a case should be instituted only on condition that the patient's confidence has been gained, and that he is willing to follow out instructions faithfully. In some instances a short vacation may suffice, or even a continuance of ordinary occupation with diminution in the amount of work, along with improved hygiene. An increased amount of rest, regularity in meals, sufficient sleep, an abundance of fresh air, mild exercise, attention to the bowels, and a cold sponge-bath in the morning, may all be helpful. Sometimes the physician does well to furnish a written schedule, outlining the way in which all the hours of the day are to be spent. Women often do well under the modified rest treatment here suggested, and many of them, even among the poorer classes, find it possible to arrange their time according to a program made out by the physician. In certain instances it may be wise to have the patient removed to the home of a relative, or a friend, who is able, and willing, to aid in carrying out directions. It is possible to make use of this method of treating patients in a neurological out-patient department. This treatment has been shown by experience to be very satisfactory as a form of therapy, and has also served as a helpful means of instructing medical students. Thus, after a patient has been established in her new surroundings, some one among the third-year students who is interested in this class of patients may be selected to take charge of the case, and to see that instructions are carried out; the student's visits and the management of the patient must, of course, be under the supervision of a member of the dispensary staff. There are few circumstances, perhaps, more prone to shake a student's faith in the efficacy of therapeutics than the witnessing of ordinary "dispensary treatment," and its utter futility, in the majority of the neurasthenic and psychasthenic states. On the other hand, the results obtainable by the method referred to above, which insures personal contact and continued observation under more favorable conditions, are most gratifying to patient, student, and instructor.

Where financial difficulties are less, many adaptations, and modifications, of partial rest treatments will suggest themselves to the thoughtful physician. Of course, in cases where expense is a minor consideration, all measures which add to comfort, and facilitate cure, may be employed.

Continuance of Occupation.—Whether a patient's occupation shall be temporarily suspended, or not, is an important matter; a decision is not always easy, requiring, as it does, the closest scrutiny of individual characteristics, and mode of life. Though no generally applicable rule can be laid down it may be worth while to refer to certain general principles which may, on occasion, be helpful in determining the course to be adopted.

(1) If the particular form of occupation be an etiological factor in the neurosis, the work may not only have to be suspended, but, perhaps, have to be abandoned altogether; this is especially true if his occupation expose the patient to some chronic form of intoxication. There are, too, certain intellectual pursuits, and professional vocations, which appear to be incompatible with the welfare of individual neuropaths. Medical students and physicians, on discontinuing their work temporarily owing to imperative ideas or fears, sometimes find, upon resuming it, that the obsessions, phobias, or impulses are so intimately connected with certain phases of their work, that resumption is impossible. We recall, in particular, the case of a young surgeon, intellectual and practically skillful, who, very early in his career, was forced to discontinue his operative work because of an absurd, but strong, impulse to spit into the wound just as an operation was being completed. Fortunately, he never yielded to the impulse, but it was so disturbing that he chose another form of medical work and, we are told, is making some progress.

(2) Not only should the character and etiological bearing of the occupation be regarded, but, what is equally important, the manner in which the work is performed. Some persons do not know how to work properly. Many are spasmodic, irregular, temporarily extremely intensive, actually going on an intellectual, or occupational, "spree" of several days' or weeks' duration. It is necessary to teach these to perform the day's duty well, to adopt the maxim of Wilhelm Meister and "do the thing just ahead," and to leave to-morrow to take care of itself. There are others who, although occupying some minor position, overestimate their responsibility, and feel that the larger problems and destinies of their employers are entirely within their keeping. This attitude is sometimes a symptom, a form of "over-conscientiousness," but it may also pertain to one particular form of occupation, so that both the symptom, and the occupation, need attention.

(3) The third general consideration regarding continuance of occupation has to do with the patient, the severity of his symptoms, and the degree of fatigue and exhaustion manifested. As Forchheimer, Savage,

Ziemssen (38), and others have emphasized, it may not be necessary in mild, or moderately severe, cases to discontinue the occupation. It will be well to have the patient take a brief rest, completely away from business or, if this be impracticable, to spend the week-ends away from home and business affairs. In most cases it is, however, necessary rigidly to limit the amount of work, and to prevent work at full speed. It is, moreover, essential to see to it that the leisure is spent in a profitable, hygienic manner, preferably in some form of outdoor exercise. Riding, hunting, walking, golf, and gymnastics may be recommended. If insomnia and incapacity for work are prominent symptoms, discontinuance of the occupation, at least for a time, can scarcely be avoided.

Requirements in the Physician.—It has been stated that a physician's success in general medical practice depends largely upon his ability to treat, successfully, patients suffering from neurasthenic and psychasthenic states. There is much truth in this saying. For, in the first place, a large percentage of every general practitioner's following is made up of this class of patients, and, in the second place, the faculties which insure success in the treatment of neurotics are of great help, also, in the management of many distinctly organic diseases. One attribute, apparently essential in physicians who have this success, is the quality of deriving genuine pleasure from the work of restoring such patients to health. If the physician is not interested in neurology, and especially if he be impatient with the complaints of the functionally nervous, he will do well frankly to confess the aversion to himself, and refuse to assume the responsibilities of treatment of this class. How frequently the remark is made, "I do not understand how Dr. So and So can spend his time fooling with nervous patients." Without interest in, or understanding of, nervous patients success in their treatment is scarcely conceivable; thus approaching them a physician may fail really before he has commenced.

The physician who will work successfully among the functionally nervous should be broadly educated, refined, sincere, honest, kind, firm, and adaptable. Whatever the patient's age, station, or race, the physician should be able to see things from his point of view, to put himself, to a certain extent, in his place, and to command his respect and confidence. Not every one possesses the kind of personal magnetism, which makes the patient willing to lay bare before him the innermost secrets of an unhappy life. Likes and dislikes, personal attractions, and repulsions, depend upon a whole series of elements in the personality; they are often instinctive—matters of first impression. Some persons immediately appeal to us, some make little or no impression upon us; still others at once excite in us a feeling of aversion. Generally speaking, one is more apt to be successful with a patient whose social condition, environment, and habits of thought and life belong to a circle not too remote from that in which one lives and moves one's self. The first essential is that the physician gain the patient's confidence; if this cannot be secured, it may be better not to assume the responsibility of the treatment. For securing the necessary mental relationship, Bouveret emphasizes (7): (1) a thorough examination; (2) a real interest in the patient's suffering; (3) an intelligible explanation of the nature of his malady; (4) repeated reassurance and prospect of cure; (5) a relation of the success that has attended the treatment of other similar patients—where possible, selecting, as examples, instances in which the symptoms were still more severe; and (6) never making the mistake, when a patient is ill, of giving him the impression that you do not believe his suffering to be real.

Confidence once established, care should be taken that it be not destroyed. Occasionally a physician's resources are taxed to the utmost. If the patient be educated, engaged in some intellectual pursuit, or have some knowledge of affairs psychical, it requires most delicate tact, the guarded use of language, apt resourcefulness, and, above all, unswerving honesty, to maintain intellectual and moral control. Once a patient detects the physician in error, or hears conflicting statements concerning his condition, he is likely to be shaken in his faith. He must be made constantly to feel that the physician knows more about his condition than he. This faith-attitude is not always easy to retain, for a patient feels that his physician has never seen anyone suffer as he does. How can anyone know more about a state which he has never experienced than one actually suffering from it? is the question which is often asked.

After a regimen has been decided upon, definite, positive, and accurate directions should be given, and, unless there be positive indications for changing them, these should be rigorously enforced. Firmness, judicious sympathy, kindness, patience, and optimism are needed in the physician, if the patient is properly to regard his instructions. Over-solicitude should be avoided; ridicule and sarcasm are almost never necessary. Further, once a psychical state has been carefully explained to the patient, the physician does well, as a rule, to decline to discuss it at great length. It is, moreover, not always wise to greet the patient, every time one enters the room, with "How are you feeling to-day?"; it is often more helpful to teach the patient to ignore symptoms as much as possible; cheerfulness and hope should be inspired.

Requirements in the Nurse.—Those patients who require the special services of a trained nurse should have the attention of one suited to the needs of the individual, not of one chosen at random. In general, the requisites of a good nurse for the care of neurasthenics are, in a measure, similar to those of the physician. The nurse should be cheerful, personally attractive, absolutely cleanly, neat, patient, and tactful, and she should have plenty of good common sense. Nurses who have had merely a general hospital training may not be entirely satisfactory, for such nurses, accustomed to the care of surgical or "acute" medical cases, have had, as a rule, but little experience with neurotic patients, and cannot tolerate their apparently absurd fancies and ideas. Nurses, like doctors, often exhibit a preference for the care of certain classes of patients; one should be selected who is interested in the care of, and the study of, neurasthenic states. It is, of course, essential that physician and nurse loyally coöperate in the maintenance of the regimen; the nurse should have respect for the physician in charge, and under no circumstances belittle his opinion, or contradict his statements, to the patient. The nurse should sedulously avoid giving the impression that any of the "discipline" originates with her; the "rules and regulations" will be submitted to more readily when they are understood to be the physician's orders. A nurse's attitude toward a nervous patient should always be one of friendliness and helpfulness; above all, it is desirable that she herself be free from any neurotic tendencies; that she be thoroughly healthy in body and in mind. If the case be a protracted one, it may be advisable to change the nurse occasionally, even when no incompatibility exists. Thrown together so intimately and continuously, mutual boredom is not surprising; moreover, the strain on the nurse is often too great to justify a continuance with one patient over a long period.

Where the physician notes an incompatibility of temperament in nurse and patient, he may change the nurse promptly; trained nurses now understand that this brings no discredit or censure with it.

Generally Accepted Therapeutic Principles

According to prevailing medical opinion, neurasthenia and psychasthenia, aside from their primary causes, are looked upon as conditions of "irritable weakness" of the central nervous system. Fatigue and irritability are both prominent features, and they furnish, when wise attention is paid also to causes, a rational basis for therapy. Both fatigue and irritability suggest the need of *rest*—the essential element in all therapeutic courses prescribed for patients suffering from neurasthenia or psychasthenia. But continued rest means inactivity, and inactivity, if too prolonged, means deterioration of function. A fatigued muscle requires rest, and a rested muscle needs exercise. So it is with the nervous system. The treatment of these neuroses will, therefore, be considered in two distinct sections: First, a section dealing with the *protection* of the central nervous system, or *sedative* treatment; and, second, one dealing with *exertion* of the central nervous system, or *stimulating* treatment. Certain more special therapeutic features, such as treatment in sanatoria, or in hospitals, by travel, by climate, etc., as well as the treatment of particular symptoms, will be separately discussed.

Protection and Reconstruction of the Central Nervous System

Rest.—Though rest is generally accepted as an essential therapeutic agent for all patients suffering from neurasthenic states, opinions differ as to the degree of rest, the length of time required, and the method of administering it. Either mental or physical rest, or both, may be prescribed, and the rest may be partial or complete.

The value of rest as a therapeutic measure was mentioned by Beard in his first communication on neurasthenia; its real function in the treatment of the neuroses was not generally appreciated, however, until Dr. S. Weir Mitchell (33) made known his method of treating these patients, and showed the world what could be done by means of systematic "rest cures." He recognized fully that rest alone was not sufficient. It tends to lessen the appetite and digestion; it may enfeeble the circulation; it induces constipation. For these reasons massage and a suitable dietary form a part of every "complete" rest treatment, and, as soon as possible, the principle of rest or protection is made to give way gradually to the principle of exertion or stimulation.

Partial Rest.—Partial rest may be variously adapted to suit individual needs, and is most useful in treating the milder conditions, as well as in ameliorating the symptoms of those who, though ill enough to justify it, cannot afford to discontinue all of their regular work. Such partial rest may vary all the way from a slight repose of an hour or so each day to a more exacting prescription to spend the greater part of the twenty-four hours in bed. The duration of and the hours selected for rest have sometimes to be adjusted to the requirements of some other obligatory routine. In many mild cases it may suffice if the patient retire an hour or two earlier than his wont, if he have his breakfast served in bed, and if he recline upon a lounge for half an hour before and after each meal. Usually it is well to have such a patient spend at first ten or eleven hours of the twenty-four in bed.

Absolute Rest.—By absolute rest is meant as complete rest of both body and mind as is possible; it necessitates, for a patient yielding to it, a more or less prolonged stay in bed. In the severest cases the patient may not be permitted to feed himself nor to rise even for urination and defecation. If complete mental rest is necessary also, isolation of the patient becomes necessary; all communication with family and friends may be temporarily cut off, and reading and unnecessary conversation for a time prohibited. These extreme measures, however, are used only in the severest cases; even where the symptoms are marked, most physicians of experience prefer some modification of a complete rest treatment rather than the unqualified edict.

Rest in bed has for its object two distinct aims: (1) to reduce physiological expenditure of energy to a minimum, and to permit the restoration of normal function in fatigued tissues; (2) to help gain the patient's confidence and to secure proper regard for the physician's instructions; in other words, to establish at the outset what is necessary for cure, namely, "medical obedience." Rest alone is often sufficient to relieve much of the feeling of exhaustion; along with suitable diet, it is a help in making undernourished patients gain in weight. One should not, however, be led to think that, because a patient puts on fat, he is necessarily gaining in strength and in muscle tissue; we do not wish to make either obese people or athletes out of our neurotic patients, but rather to put all the tissues into a healthy state; this is why, after preliminary rest and abundant feeding, we have, later on, to consider most carefully the matters of exercise, and of suitable balance in the diet of protein, carbohydrate, fats, salts, and water.

The bed should not have a feather mattress, should be inviting, clean, of moderate firmness. The room should be well ventilated, the clothing light and suitable for the season and temperature. If the bed can be run out on a porch in fine weather, all the better.

Rest in bed is clearly indicated for all patients who show real exhaustion or evidence of marked malnutrition. Extreme irritability and emotional outbreaks are also indications for rest. Bouveret (7) advises rest in cases of cerebrasthenia, and more particularly in those of myelasthenia, where backache, pains in the extremities, and gastric symptoms are pronounced. Ziemssen (38), Buckley (9), and Godlewski (20) all suggest that, in the cases with "wornout" minds without marked somatic signs, and especially the fat, robust-looking, normally digesting patients with neurasthenic complaints, rest in bed is rarely indicated, or, if at all, only partial rest for a time. Charcot's type of patient, which he characterizes as *l'homme aux petits papiers,* will also do better with only moderate rest, combined with a prescribed routine of judicious exertion.

The duration of rest, the time of year best suited for it, and the degree of it are all largely matters for individualization. A rest cure is most easily carried out in the cooler months of the autumn or spring. When rest has been decided upon, the maximal rest and the more rigid restrictions to be used in a given case should be given at the very beginning of the treatment. It is better, and far easier, to reduce the rigor of one's rules after a time than it is to increase it. It makes a better impression upon the patient, too, for an increase of liberty encourages the idea of improvement; the "conviction of cure" is strengthened when some of the bars can be let down because of progress. The duration of the rest necessary will depend largely upon the actual progress the patient makes; no definite time should be set at the beginning, or, if any "guess" is made, it should err on the side of overestimation. Some patients will not require more than two, four, or six weeks; in very severe cases months may be necessary.

Isolation.—Like rest, isolation may be partial or complete. It has for its main object the removal of the patient, as far as possible, from all sources of external irritation; it serves also to increase confidence, and to make the physician's control over the case more easily possible. In extremely irritable and hypersensitive individuals, upon whom even normal external stimuli, owing to distorted perception and abnormal association, yield exaggerated reactions, there may even be a personal desire for separation from the external world; temporary isolation is for them a real relief. Though the majority, perhaps, of patients may be successfully treated without isolation, there are some in whom therapy will almost surely fail, unless the patient can be wholly separated from his or her ordinary surroundings and associates.

Partial isolation away from home should be prescribed in most cases where rest in bed is indicated. Separation from overanxious and oversympathetic or undersympathetic friends and relatives is most essential. Many neurotic patients feel that their complaints are misunderstood by the family, that they are neglected, or even abused. And it is not uncommon actually to find patients reproved, scolded, and censured for symptoms which they are wholly unable to suppress, or in other instances to see patients humored, encouraged, and excused from all responsibility—to their detriment.

The need of isolation, then, depends somewhat upon the conditions which exist in the family circle. If it be thought necessary to insist upon it, the physician, while it is being carried out, should not forget that a large share of his work consists in reëducating, not only the patient, but also other members of the family; he must work for a readjustment of conditions in the home.

Letter-writing between patient and family may have to be largely restricted, or even forbidden altogether at first, though the arrangement should always be made that, if anything goes wrong at home which the patient really should know, knowledge of it will not be withheld. Visitors may later on be permitted once a week for a short time only, provided they have a proper understanding of the patient's condition. It should be remembered that a single visit, by an ill-chosen person, may undo a week's work.

In some few cases, isolation may be undertaken at home, but with the distinct understanding that, in addition to the nurse, only one member of the family, or one attendant, chosen by the physician, is to have access to the patient's room. Isolation without a nurse or a companion would in most cases do more harm than good.

Absolute isolation with a nurse is, indeed, a trying ordeal, and should be reserved for the severer cases, in which the emaciation, irritability, or hypersensitiveness are pronounced enough to demand radical measures.

In some psychasthenic states, it is better not to isolate the patient, particularly if it seem probable that it will encourage introspection, self-analysis, or despondency. When convinced, however, that isolation, partial or complete, is necessary, we should not be deterred from prescribing it by the patient's objections, or by the statement that she could not endure separation from family and friends. A patient can usually be made to see the wisdom of isolation, when it is needed, by a few well-chosen, kind remarks, the physician giving the reasons first, and commenting upon the efficacy of isolation in the treatment of similar cases in his experience.

Diet.—Diet in the treatment of neurasthenic states has been the subject of much discussion; many fanciful dietetic measures founded upon various conceptions of the pathology of these states have been advocated. Autotoxemia from the gastrointestinal tract, changes in vascular tension, gout, and disordered metabolic states, including the *arthritisme* of the French school, all have had a part in influencing dietetic régimes. It would be just as irrational to formulate a specific diet for all neurotic patients as it would be to treat all cases of headache in the same way. Whatever may be one's view as to the etiology and pathology of psychoneurotic states, diet should be prescribed according to the individual requirements of the patients. In general, on the nutritional side, neurotic patients may be divided into two classes: (1) the lean, emaciated, underfed, so-called "wornout" class, and (2) the healthy-looking, fat, ruddy, truly irritable type. French observers, especially, have supported this classification, recognizing two main types, on the basis of a study of vascular tension and of analyses of the gastric juice. Members of the first group usually exhibit arterial hypotension, atony of the gastrointestinal tract, and hypoacidity or even anacidity of the gastric secretion. In the second class arterial hypertension, gastrointestinal restlessness, and hyperacidity of the gastric juice are often demonstrable. Although some who thus divide the cases are ardent supporters of the "rheumatic" nature of the neuroses, and have established dietetic régimes largely based upon this belief, this fallacy should not deter us from recognizing the usefulness of their observations upon blood pressure and gastric function as helpful guides for prescribing dietetic measures. As far as our own studies upon the relation of vascular tension and gastric acidity to the two types go, they support the classification.

Dietetic measures may be instituted to secure physiological rest of the alimentary tract; to adjust the food intake to certain abnormalities of the metabolic processes, and to regulate definite gastrointestinal functions. In selecting a dietetic régime we should have in mind the state of nutrition of the patient, any gastric disorder present, as shown by a study of the gastric juice and of the motility, and the blood pressure. Special indications for dietetic supervision include evidences of malnutrition, anemia, digestive disturbances, irregular or faulty habits of eating, metabolic diseases. As a rule, the fat, normally digesting cerebrasthenic requires little attention to diet, other than measures suited to his vascular tension, or to reduction of the body weight. Often the alimentary supervision in neurasthenic states is less a matter of specific dietary, or departure from what a healthy individual should observe, than an effort to reëstablish rational eating, educating the patient to enjoy normal amounts of the ingredients of any well-selected menu.

Diet may be quantitatively restricted in calories, or it may be qualitatively altered. Quantitatively, it may be temporarily reduced to a minimum, or it may be increased beyond the limits ordinarily required to maintain metabolic equilibrium, as in "forced feeding" or "superalimentation." Both of these methods may be, and usually are, employed at different stages in the treatment of a single case. Thus a restricted diet is used when we wish temporarily to secure digestive repose; in such cases it is common to begin with small quantities of milk, given often, without other food, for a few days or a week. In cases with marked gastrointestinal atony, dilatation, and diminished gastric secretion, some care must be exercised in giving milk lest fluids further dilute an already impoverished gastric juice, favor gastric dilatation, cause diarrhea or constipation, and really starve the patient. In such cases it may be wiser to employ a mixed diet, moderately restricted at first, especially as to fluids, only small quantities of water being allowed with meals. Even though, as Hawk has recently pointed out, the ferments act better when water is taken with meals, still in gastric atony large quantities of water interfere with gastric motility. When gastrointestinal symptoms are absent, or of minor importance, except for anorexia, the gastric juice being normal, certainly no harm, but distinct benefit, may be derived from a brief course of milk feeding. Ordinary milk may be given cold, or it may be given skimmed, boiled, or mixed with lime water. Patients often have a distaste for milk, asserting that they "never could drink milk"; then a little cocoa, tea, or coffee may be added, just sufficient to color it, if desired, though usually, on gentle persuasion, patients will take it plain, and, resting, find that they can digest it.

This preliminary "starvation diet" has the support of such men as S. Weir Mitchell (31), Dubois (17), Playfair (37), Allbutt (1), Starr, and others. Our own experience has led us to adopt it for a few days at the beginning of treatment in the majority of cases. It has seemed to us that patients gain more rapidly later, on account of it. Godlewski recommends it most heartily in cases with arterial hypertension. Our own experience has confirmed his statement that a period of only two or three days may suffice to cause a marked fall in blood pressure. The empirical use of a preliminary milk diet as a rigid routine in certain institutional treatments, for every case admitted, is, of course, to be deprecated; but, judiciously employed, after careful study of the individual patient, it has its place, and should, in suitable cases, be prescribed without hesitation.

After the preliminary period of restriction, a general mixed diet may usually be given. It should be appetizing, sufficient to maintain nitrogenous equilibrium, easily digested, and it should contain proper proportions of the essential elements—proteins, fats, carbohydrates, and salts. If the patient be obviously underfed, an attempt should be made to fatten him; if he be wilfully overindulging himself in food, we regulate the amount, and reduce his weight. In the latter case Buckley (9) advises the "Salisbury method," restricting the diet, for a time, to rump steak, cod fish, and hot water. The absence of carbohydrate, fats, and fruit from this diet makes it objectionable. It is better to use one of the diets for obesity (q. v.). The patient should be weighed accurately once a week, and a weight chart should be kept. Godlewski, paying much attention to the blood pressure in cases of hyperarterial tension, hyperchlorhydria, and motor restlessness, advises a restricted dietary, eaten alone, all external stimuli likely to cause reflex psychical irritation of the alimentary tract being reduced to a minimum. In hypochlorhydria with gastric atony and low pressure he advises meals with others, with plenty of psychic stimuli to the gastric secretions (table decorated; appetizing dishes). The company should be cheerful and agreeable, and all work should cease an hour before each meal. Déjerine requires this class of patients to eat under the direction of, and in the presence of, an attending physician who supervises the meal. Sir Andrew Clark emphasized eating slowly; "the mouth having thirty-two teeth, each mouthful should receive thirty-two bites." Fletcherism has had some vogue among neurasthenics, but excessive "bradyphagia" is not to be encouraged.

Forced feeding, after preliminary rest and preparation of the stomach, is, as has been said, particularly applicable to the thin, anemic, underfed type of patient. The principal point in superalimentation is *frequent* feeding, including, besides three well-appointed meals a day, an early morning and a late evening repast, and interim meals of eggs, crackers, or milk. Forced feeding is often used for too long a period, after it has ceased to be beneficial. In cases of undernutrition, when judiciously managed, for a proper length of time, it is of signal benefit. It is manifestly inappropriate for the robust, healthy-looking neurotic, already overburdened by excessive assimilation from a constantly overindulged stomach.

Qualitative dietetic restrictions, necessary in the neurasthenic states accompanying gout, diabetes, etc., can only be mentioned here. For the details the reader is referred to the special chapters dealing with these subjects. A few remarks bearing upon the relative proportions of the main constituents of dietaries in neurotic cases may be of help; comments upon some typical dietetic schedules which have been recommended also here find a place.

Proteins.—The general opinion has already been expressed that excess of proteins, of meats in particular, should be avoided. Bouveret advises that, in all cases with diminished hydrochloric acid in the gastric juice, highly seasoned meats should be prohibited. Collineau, who adopts the "arthritic" theory of neurasthenia, feels that the proteins should be reduced. Combe, of Lausanne, has advocated a diet, widely used upon the Continent, which is entirely meat-free. He designates it as a "farinaceous diet without meat," and suggests the following schedule:

7.30 A. M.—Thick soup, water, milk, biscuit, and butter.
10.00 A. M.—Farina with milk.
12.30 P. M.—Yolk of one or two eggs, *pâté alimentaire,*[1] purée of potatoes, pudding, toast, or biscuit, and butter. No water.
3.30 P. M.—Farina with milk.
7.00 P. M.—Same as at 12.30.
10.00 P. M.—Évian water; after ten days a baked potato is added.

This schedule is continued for from three to six months; during the treatment his patients, he asserts, show marked improvement.

Fats and Carbohydrates.—Beard advised the reduction of starches and sugars, but fed fats, oils, butter, and milk generously. Dana advises plenty of fats and nitrogenous foods, but is opposed in general to the establishment of any special dietetic régime. The farinaceous diet of Combe has been referred to above. General opinion favors a mixed diet for most cases, with a preference for quantitative rather than qualitative changes. In arterial hypertension with hyperchloridia, Godlewski advises a milk and egg salt-free diet with very little water during meals. Milk or mineral waters, however, may be given between meals; it is well in most cases, for a time at least, to prohibit tea and coffee.

In making a schedule we should remember that neurotic patients, self-centered, apprehensive, and suggestible, are often afraid to eat; the details of an elaborately prepared dietetic schedule may only serve to fix their attention upon the alimentary tract and to increase a chronic dyspeptic invalidism. *As soon as possible the patients should be taught to eat sensibly of all easily digestible foods, regardless of inclination.*

The Weir Mitchell Method.—One of the most valuable protective measures we may employ is the course of therapy advised by Dr. S. Weir Mitchell (33) of Philadelphia, whose name has become inseparably associated with the "rest cure" treatment of neurasthenic and hysterical conditions. The essential features of the method are: rest, isolation, and diet (superalimentation), with massage and electricity to promote circulatory and muscular activity. It has been especially useful for neurasthenics who are anemic and emaciated; for these "fat and blood" must be made. As a rule, the more emaciated the patient, the easier he is to treat. In the severest cases, the patient is put to bed, made to rest absolutely in isolation, under the care of a nurse, preferably in some country district. Usually she is not permitted to receive, or to write, letters; even self-feeding may be prohibited. The patient is not permitted to speak of her ills to any one except the physician; reading and conversation are not allowed for a time. The diet, in the beginning, consists entirely of milk, skimmed, or peptonized, if necessary, or diluted with plain, carbonated, or lime water. For the first seven days one quart of milk is administered in the twenty-four hours. Thus, beginning at 7 A. M. and ending at 9 P. M., four ounces of milk are given every two hours; it is advised that it be slowly sipped. During the second week the total amount of milk in the twenty-four hours is increased to two quarts. Later, a light breakfast is added, and, within the following ten days, three full meals are permitted, with milk between the principal repasts.

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[1] *Pâté alimentaire,* made of milk and flour and cooked for twenty or forty minutes in salt water.

Massage is usually begun on the fourth day, and is given gently, in the following order: feet, legs, back, chest, and abdomen; twenty minutes' duration in all. Within a week, the massage is given for one hour daily. Tapping and slapping are to be avoided, the massage consisting rather of stroking, kneading, and gentle rubbing. Electricity may be given along with massage, or may alternate with it. The induced current is applied to the spine and to the general musculature for ten or fifteen minutes daily. If any of the measures are to be dispensed with, electricity may be most readily omitted. After the stage of three full meals has been reached, Swedish movements may be commenced. In some cases, cod-liver oil, a little wine, or iron and strychnin tonics may be administered. In Weir Mitchell's hands remarkable cures have been obtained; no small part of his success lay doubtless (1) in his wise individualization of the treatment, and (2) in the accompanying psychotherapy, for which the richness of his personality made him unusually well fitted.

Weir Mitchell's method has been employed all over the world, notably by Playfair in England and by Binswanger in Germany. Many modifications and adaptations have been devised. Sample schedules, arranged by Binswanger, by J. K. Mitchell (son of Weir Mitchell), and by M. Allen Starr follow. Excellent accounts of the rest cure, by one who has successfully applied it in a large and varied experience, are available in the articles by F. X. Dercum in the "Physiological Therapeutics" of Solis Cohen (16), and in Musser and Kelly's "Handbook of Treatment."

Feeding in Rest Cure Cases According to Binswanger

7.00 A. M.—Glass (250 c. c.) boiled milk, or cocoa made with half milk and half water; two or three biscuits or zwieback.
9.00 A. M.—Cup of bouillon; 2/3 oz. (20 gms.) meat; 1 oz. (30 gms.) Graham bread or toast; 1/3 oz. (10 gms.) butter.
11.00 A. M.—4½ oz. to 6 oz. (125-175 c. c.) milk, with a tablespoonful of meat extract, or the yolk of an egg.
1.00 P. M.—2½ to 3½ oz. (80-100 c. c.) of soup with oatmeal, barley, or rice; 1¾ oz. (50 gms.) roast; 1/3 oz. (10 gms.) potatoes; ¼ to 1/3 oz. (7-10 gms.) vegetables; 2/3 oz. (20 gms.) sweet rice pudding; 1¾ oz. (50 gms.) stewed fruit.
4.00 P. M.—4½ oz. (125 c. c.) weak tea, coffee, or malted milk and two biscuits.
6.00 P. M.—2/3 oz. (20 gms.) of meat, which may be hot or cold roast, scraped raw meat, tongue, or ham; 1/3 oz. (10 gms.) Graham bread, or toast; 1/6 oz. (5 gms.) butter.
8.00 P. M.—4½ oz. (125 c. c.) soup, cooked with 1/3 oz. (10 gms.) butter and the yolk of an egg; oatmeal, barley, rice, etc.
9.30 P. M.—4½ oz. (125 c. c.) malted milk.

These quantities are gradually increased until, by the end of two weeks, the amounts of milk, cocoa, and soup are doubled, and those of meat, bread, and butter trebled. Small quantities of fresh vegetables and simple puddings are then allowed. There are many cases in which such a menu can be prescribed from the beginning of treatment, particularly in the cerebral type of neurasthenia without marked emaciation or without marked gastrointestinal disturbances.

Sample Full Rest Schedule According to J. K. Mitchell:

7.00 A. M.—Cocoa, cool sponge bath with rough rub, and toilet for the day.
8.00 A. M.—Breakfast, with milk. Rest an hour after.
10.00 A. M.—Peptonized milk, 8 oz. (236 c. c.).
11.00 A. M.—Massage.
12.00 M.—Milk or soup, 8 oz. (236 c. c.). Reading aloud by nurse, half hour.
1.30 P. M.—Dinner. Rest an hour.
3.30 P. M.—Peptonized milk, 8 oz. (236 c. c.).
4.00 P. M.—Electricity.

6.30 P. M.—Supper, with milk. Rest an hour after.
8.00 P. M.—Reading aloud by nurse for half an hour.
9.00 P. M.—Light rubbing by nurse with drip sheet.

In addition to the above, he advises the following: 3 oz. (98 c. c.) malt extract with meals; 8 oz. (236 c. c.) peptonized milk with a biscuit at bedtime, and a glass of milk during the night, if desired. An occasional laxative (cascara, 10 to 30 drops, 0.61 c. c.) is administered, and later Swedish movements are added, to be done after the massage.

In milder cases partial rest, diet, occupation and diversion are prescribed, and a schedule is arranged so as to occupy most of the patient's time. As a sample régime, one suggested by M. Allen Starr may be given; changes may be made to suit individual needs.

Sample Schedule for Partial Rest Cure (M. Allen Starr):

8.00 A. M.—Small cup of coffee, with hot milk; or black coffee, if preferred. Hunyadi water if needed.
8.15 A. M.—Morning toilet.
8.30 to 9.00 A. M.—Breakfast: fruit, cereal with cream, eggs, bacon or fish, hot milk or cocoa.
9.00 to 10.00 A. M.—Rest. Letters read by nurse, or patient, after 9.30; glass of water.
10.30 to 11.00 A. M.—Bath; large bath towel under patient; blanket over patient. Nurse to bathe each part with soap and tepid water, or give patient a salt rub, or pack in place of bath with salt water affusion.
11.00 to 11.30 A. M.—Glass of milk or kumyss, or hot broth, or cocoa. Rest.
11.30 A. M.—Get up and dress for the day.
12.00 M.—Drive or walk.
1.30 P. M.—Lunch: soup, steak or chops, with vegetables; salad; baked apple or fruit.
2.00 to 3.00 P. M.—Rest quietly, lying down, relaxed, but not undressed. Glass of water.
3.00 to 4.00 P. M.—Walk, drive, or see friends. Glass of milk or beef tea. Undress.
5.00 P. M.—Massage, at first gentle, later Swedish movements, or wet sheet pack, or physical culture exercises.
6.00 to 6.30 P. M.—Rest, alone, lying down.
6.30 P. M.—Dress for dinner. Glass of water.

7.00 to 8.00 P. M.—Dinner: oysters, soup, fish, game, or chicken; vegetables of any kind; salad, cheese, or fruit. No wine or coffee.
8.00 to 8.30 P. M.—Rest.
8.30 to 10.00 P. M.—Reading, or games.
10.00 P. M.—Bed, preceded by spinal douche, or drip sheet. Cascara tablet, 5 minims (0.31 c. c.) of fluid extract. Glass of hot milk, without or with trional, as required.

There is a group of still milder cases, for whom a still less rigid régime may suffice. The easily fatigued, somewhat undernourished, apprehensive individual, who is still capable of attending to the ordinary affairs of life, though they are felt as burdensome; tired housewives, and exhausted men of business form a large contingent. They complain of loss of power of concentration, slight irritability, fickle appetite, mild gastrointestinal symptoms, and have a low blood pressure. They may begin the day with a "tired feeling," or they may work in comparative comfort until four or five o'clock in the afternoon, when symptoms of abnormal fatigue begin to appear; the end of the day finds them exhausted in mind and body. Many of them cannot afford an expensive "rest cure" or even a prolonged rest at home. In case no organic disease exists, a more liberal schedule, but one which can and will be observed, may suffice for them. We have found the following very satisfactory:

7.00 A. M.—Cup of cocoa, or weak Oolong tea, with cream and sugar; or a glass of milk.
7.30 A. M.—Rise; 5 or 10 minutes' exercise with dumbbells, Indian clubs, or Whitely exerciser; or, still better, the exercises outlined in J. P. Müller's "My System" (New York, G. E. Stechert & Co.). Cold sponge bath, 70° F (21° C.), followed by brisk rub with coarse towel.
8.00 A. M.—Breakfast: mixed diet, followed by quiet reading of mail or papers.
9.00 A. M.—Customary occupation for the day.
11.00 A. M.—Glass of milk, 8 oz. (236 c. c.), and cracker.
1.00 P. M.—Lunch: not to be a "quick lunch," but a liberal meal, served at a table, preferably in the company of friends, and eaten slowly, during one hour, away from business.
4.00 P. M.—Glass of milk, 8 oz. (236 c. c.), and cracker.
5.00 to 6.00 P. M.—Riding, driving, walking, or gymnasium.
6.00 to 6.45 P. M.—Rest on lounge or bed.
7.00 P. M.—Supper; no tea or coffee.

9.30 P. M.—Warm bath, 95° F. (35° C.). Glass of hot milk and cracker.
10.00 P. M.—Retire.

A general tonic may be given (especially if the appetite be poor), and an occasional dose of cascara (M. v to xx—0.3 to 1.0 gm.—of the fluid extract) at bedtime. Massage and mild hydrotherapeutic measures may with advantage be introduced into the schedule in some cases.

Should the "rest cure" be decided upon, it is better, as has been pointed out above, to prescribe it *ad maximum* at the start, with all its essential components, and gradually to relax the rules rather than to approach it by degrees, since unsuccessful attempts with partial rest are apt to shake the patient's confidence in more restrictive measures. It should always be remembered that, in the "rest cure," physical measures are not *all* that is needed; the personalities of the physician, nurse, and masseuse are important; no doubt, in some of the instances in which "rest cures" have failed, negligence on either the physical or psychical side has been responsible. Further, some of the cases, taken to be "neurasthenia" at first, and which do not respond to a well-ordered "rest cure," doubtless turn out on longer observation to be either early stages of serious psychoses, or states symptomatic of organic disease.

The Dubois Method.—While followers of Weir Mitchell have sometimes laid the main emphasis upon the physical effects of rest, isolation, and forced feeding, the founder of the method paid much attention also to the psychic side of his cases. Attention to the latter has, since 1904, when Paul Dubois, of Berne, published his experiences in "The Psychic Treatment of Nervous Disorders" (17), become more general. Psychotherapy has been used indiscriminately by the charlatan, the faith healer, and the fakir from time immemorial; qualified physicians have also long used psychotherapy, sometimes consciously, sometimes unconsciously; Dubois is one of those who have tried to establish its use on a solid basis. After having employed the Weir Mitchell method of treatment for a period of twenty years, he gradually came to attach less importance to the purely physical features of rest, isolation, and overfeeding, and to regard these measures more as a means of securing a receptive psychical attitude in the patient. He modified gradually the degree of isolation and rest, gave up the use of massage and electricity, and employed vigorous psychic treatment in the form especially of persuasion and argumentation. Dubois is a believer in "determinism." The "will" is for him a product of hereditary endowment, education, and environment. Men are able, when taught how, to work toward ethical perfection. His motto is: "Gain insight, and strengthen the will, and you will be happy."

In the neurotic patient Dubois sees an abnormal mental state, due to faulty character, expressing itself in phobias, asthenia, depression, or hypochondriacal symptoms. Fear and cowardice are, for him, states to be surmounted. The therapy consists in ethical development, in the strengthening of the will and of the character. He depends chiefly upon bringing conviction of this to the mind of the patient. By means of an ethical transvaluation, the patient regains his self-confidence and his energy; by a sort of "moral orthopedics" he becomes cured of his neurosis!

Dubois finds a gradually increased milk diet *at the beginning of the treatment* a valuable accessory; we have used this part of his treatment frequently, and can speak most highly of it.

Milk Diet According to Dubois.—The figures in the table refer to the "doses" of milk. One dose equals 3 oz. (88.71 c. c.):

	Hours of Day.								Total Amount In 24 Hours
	7 A.M.	9 A.M	11 A.M.	1 P.M.	3 P.M.	5 P.M.	7 P.M.	9 P.M.	
First Day....	1	1	1	1	1	1	1	1	24 ozs. 709 c. c.
Second Day..	1½	1½	1½	1½	1½	1½	1½	1½	36 ozs. 1,064 c. c.
Third Day....	2	2	2	2	2	2	2	2	48 ozs. 1,419 c. c.
Fourth Day..	3	2	2	3	2	2	3	2	57 ozs. 1,685 c. c.
Fifth Day....	4	2	2	3	2	2	3	2	60 ozs. 1,774 c. c.
Sixth Day....	4	2	2	3	2	2	3	2	60 ozs. 1,774 c. c.

The hours of the three future meals are marked by the larger quantities of milk at seven, one, and seven o'clock.

On the seventh day the regimen changes abruptly, and without transition he prescribes as follows:

7.00 A. M.—Breakfast: milk, 12 oz. (774 c. c.); bread, butter, honey, or preserves.
10.00 A. M.—Milk, 8 oz. (236 c. c.).
1.00 P. M.—Lunch or dinner, a full meal, without permitting any choice. This should be varied and copious, but without wine.
4.00 P. M.—Milk, 8 oz. (236 c. c.).
7.00 P. M.—Dinner or supper, which should be equally copious.
9.00 P. M.—Milk, 8 oz. (236 c. c.).

Dubois states that the effect of this treatment varies according to the case. Those who have not been copious eaters, and who are extremely emaciated, may show some gain in weight during the first week. Patients, however, who have been large eaters usually lose some weight. At the end of the second week both types begin to show a decided increase in weight, varying from two to ten pounds. This, in itself, often brings with it a feeling of euphoria.

Massage.—In all patients undergoing full rest treatment, massage is a desirable accessory measure. In milder cases it is often a helpful adjuvant. Massage may be general or local, and may be used so as to produce either soothing or stimulating effects. In the neuroses it is customary to employ general massage, and to use at first only those movements which have a sedative influence.

The effects of massage are, in its milder application, distinctly soothing upon the central nervous system, while at the same time it stimulates the flow of blood and lymph, furnishes gentle exercise to the muscles, stimulates cutaneous activity, causes an increase in the number of red blood corpuscles, and produces a decided psychical reaction. The choice of a masseur or masseuse is a matter of importance. The attendant should be refined, modest, gentle, and of pleasing appearance, and he (or she) should possess some knowledge of the neurasthenic eccentricities. In case the choice has not been well made, it is wise to change; for a psychical effect, when it is not helpful, may be detrimental. Local massage, because of its tendency to fix the patient's mind upon a particular region, should be cautiously prescribed, if at all. General massage may in the beginning aggravate the symptoms somewhat and disturb sleep; but this effect is only temporary, as a rule, and should not lead one to discontinue it. When the neurasthenic state is associated with organic disease, certain parts of the body may have to be avoided by the masseur. Only stroking and kneading movements should be used at first for a short period; gradually the time may be increased to one hour, and in a few cases massage may be given twice daily for an hour at a time. Most patients get along well with massage three times a week. After the patient gains weight, the more vigorous methods may be used, and stimulating gymnastics or Swedish movements may be added.

Eleven o'clock in the morning or four o'clock in the afternoon are convenient hours for massage for most patients. If insomnia be a prominent symptom, massage, a half hour, or an hour, before bedtime sometimes alleviates it. Some patients are, however, made more wakeful by late massage.

Soothing Hydrotherapy.—This is one of the most valuable aids in the general and symptomatic treatment of neurasthenic states. The effects of water may be sedative or stimulating, depending largely upon temperature, duration of treatment, and method of application. Aside from their physical effects, hydriatic measures carry with them certain suggestive effects upon the psyche. In general, warm (92° to 98° F.—33.3° to 36.7° C.), tepid (85° to 92° F.—29.4° to 33.3° C.), and cool (60° to 70° F.—15.6° to 21.1° C.) applications have a soothing effect; while hot (98° to 106° F.—37.6° to 41.1 C.) and cold measures (40° to 65° F.—4.4° to 18.3° C.) are stimulating. The choice of procedure will depend, then, upon the reactive powers of the patient and upon the special effect desired. Temperatures below 85° F. (29.4° C.) when applied to the general body surface cause (1) vasoconstriction in the skin, (2) stimulation of the heat-regulating center, with subsequent peripheral dilatation, and (3) depression of the vasomotor center.

In the treatment of neurasthenic states, the mildest hydrotherapeutic measures are, as a rule, the best. Potter, Ziemssen, Bouveret, Buckley, and others warn against the use of violent stimuli, such as very cold or prolonged applications, or too vigorous friction; they may produce marked depression or actual shock. Godlewski has found the milder treatment especially efficacious in those with arterial hypertension, in whom there has followed a decided fall in blood pressure; he reserves the stimulating measures for the patients with hypotension. Routine observation of the blood pressure before and after the use of hydrotherapy are helpful as guides to treatment.

Among the soothing hydrotherapeutic procedures a few may be especially mentioned.

TEPID OR WARM SPONGE BATH.—The patient, in bed, is gently sponged from head to foot with fresh water, at a temperature of 95° F. (35° C.). The body is dried without much friction, or the sponging may be followed by an alcohol rub and rest. The sponge bath is best given in the morning.

FULL TUB BATH.—This may be given tepid or warm, the duration varying according to the temperature used. If below 90° F. (32.2° C.) it should not last longer than five or ten minutes, and should be followed by gentle friction, a warm dressing gown being provided. Shivering should shorten the stay in the tub. A warmer bath is more soothing, and may be given either in the morning or just before retiring. The latter hour is chosen when a soporific effect is desired, the bath lasting from twenty to thirty minutes. For patients upon partial rest treatment who complain of a tired feeling upon waking, a hot bath, 100° F. (37.8° C.), followed by a cool spray or sponge, is often very beneficial on rising.

WET PACK.—This is one of the most valuable of the hydrotherapeutic measures. Its effects are both stimulating and sedative; the stimulation is only temporary and is followed in a few minutes by its soothing effects. A rubber sheet, covered by a double dry blanket, is laid upon the bed. A sheet soaked in water at 85° F. (29.4° C.) is wrung as dry as possible and spread smoothly over the blanket. The patient, disrobed, is placed upon this, and the sheet is snugly wrapped about the body, between the legs and about the arms, so as to avoid air spaces, which are apt to cause chilliness and discomfort. The blanket is then wrapped about the body in a similar manner, and two additional blankets are thrown over the patient. A hot water bottle is placed at the feet, and a wet towel, wrung out of water at 95° F. (35° C.) placed upon the forehead. The pack should last twenty minutes or half an hour. Upon removal from the pack the nurse rubs dry with towels, or gives an alcohol rub. Occasionally friction is employed during the pack. Entrance into and exit from the wet sheet should be rapid, care being taken to avoid chilling. Many patients will at first object to a wet pack, but its disagreeable features, and the patient's aversion to it, are soon overcome, unless it be faultily given. The wet pack may be given daily for as long as two or three months, in which case the temperature may be reduced a degree or so every day, until 60° F. (15.6° C.) is reached. Used in the evening, it is a valuable means of overcoming insomnia.

Drip Sheet.—With the patient standing in a bath tub containing just enough hot water (100° F.—37.8° C.) to cover the ankles, a dripping wet sheet taken from water at 80° F. (26.7° C.) is thrown about the body and brisk friction with the hand over the sheet is commenced. The attendant may rub the back, while the patient rubs the chest and abdomen himself. This is continued about one minute. A warm dry sheet is next thrown about the patient and friction is applied, or he may be briskly rubbed with warm towels. A short rest after the treatment is advisable. The temperature to which the patient responds most readily can soon be ascertained. The drip sheet may be given in the morning or evening. It, too, is useful in combating insomnia.

Douches.—These may be local or general, and mild or vigorous, depending upon the force with which they are given. For sedative purposes the warm, gentle douche, of short duration, is used. A variety of applications have been found serviceable. Potter recommends the Scotch douche, or a warm douche, to the lower part of the body. Bouveret has had good results from general douching, beginning with a temperature of 76° F. (24.4° C.), and gradually reducing to 60° F. (15.6° C.). Applications to the head and neck, however, are avoided. Godlewski finds the hot douche (104° F.—40° C.), given either as a shower or jet of moderate force, very effective as a sedative. After the douche friction is applied, and a warm bathrobe put on. In the spinal type (myelasthenia) good results are often obtained by having the patient sit on the edge of a bath tub, while the attendant sponges the back, from shoulders to tip of spine, with hot water (105° F.—40.6° C.) for three minutes; this is followed first by an affusion of cold water (75° F. —23.7° C.), and then by a dry rub with a hot towel. Wood recommends a "submerged douche" as an excellent sedative. For this the patient reclines in a bath at a temperature of 93° F. (33.7° C.), and a jet of hot water at 104° F. (40° C.) from a "rose" or nozzle is directed under water upon various parts of the body. Here the psychic effect is doubtless an important feature.

Any of these procedures may be given at home, in a general hospital, or in an especially equipped institution. The sponge bath, full bath, and wet pack may, in milder cases, where removal from home is impracticable, be successfully employed at home without the aid of a trained attendant. In general, however, better results are obtained when hydrotherapy is administered in a regularly equipped sanatorium for the treatment of nervous patients. But, even in a sanatorium, no elaborate hydrotherapeutic establishment is necessary. For further details on the application of hydrotherapy to nervous cases the work of Hinsdale, or that of Kellog, may be consulted.

Sympathy and Encouragement.—The physician is fortunate who can apportion to each psychoneurotic patient the kind of sympathy he should have, in right amount. To discriminate wisely and to administer sympathy judiciously are often difficult matters. A thorough recognition of the psychical element in neurasthenic and psychasthenic states, and an understanding of the reality of mental suffering, are essential. One must try to put oneself in the patient's place. It must never be imagined that it will suffice to say to him, "There is nothing wrong with you. Your troubles are imaginary; go to work and forget them." When one can do so, it is better to say: "I have examined you thoroughly, and can find no serious organic disease anywhere. Your nervous symptoms have been making you very miserable, but you can, I believe, be relieved." On medical visits, should numerous nervous symptoms be reported, it is not always well to turn a deaf ear. One has to listen to them patiently, and not appear to be in a hurry. Usually it is a relief to the patient to learn that his symptoms are not at all uncommon in nervous cases, and that, though they cause discomfort, they are not dangerous in themselves. The patient is taught to bear them as well as he can until they pass, and is urged to ignore them as far as possible. The physician who gives a local treatment for every local symptom will, as a rule, fail to help his patient.

Encouragement must be systematically given, particularly in the cases under prolonged full rest treatment. Its necessity as a therapeutic measure is well expressed in the words of Dr. Clifford Allbutt (1), who says: "The patient who can lift his eyes to the future will recover; he whose thoughts writhe in the past is on the broad road to lunacy." Encouragement brings needed calm, and helps to give poise to apprehensive neurotics. They have been ill, perhaps, for years; they have tried many "cures" and consulted numerous physicians without relief, until, finally, their confidence in themselves, as well as in the medical profession, may have been severely shaken; often they have almost resigned themselves to chronic invalidism. If one can honestly hold out to these discouraged ones the hope of relief, if he be able to relate instances of cure in similar cases, if he take care to minimize temporary setbacks and to dwell on every sign of improvement, the patients "gather up their loins" and go forward. The physician does well to trust his patient, to make him feel that he has confidence in him. Much is gained by assuring a patient that he will often find that he can really do the thing he fears he cannot do. The physician may create a desire in the patient to fulfill expectation in the way of improvement; when this can be done the benefit is often speedily attained and surprising.

Exertion of the Central Nervous System

Gradual Return to Bodily Exercise.—Toward the close of the protective régime, stimulating and tonic measures are gradually begun. More vigorous and prolonged massage, including deep kneading and tapotement, may be used to advantage, and Swedish movements given. Active movements against gradually increased resistance may be prescribed. The patient should, after a rest cure, be allowed to get up only gradually. Where rest has been complete one begins by allowing him to sit up in bed or in a comfortable chair, preferably in the open air, for a short time each day. At first he should be up only in the mornings; later, also in the afternoons. The time up is gradually increased, until finally most of the day is spent out of bed. By this time, too, the patient has been allowed to walk a little, and the walking may be slowly increased in amount until four or five miles per day are covered. Calisthenic movements may be taken up, and later more complicated and more energetic gymnastics. It is well to furnish the patient with a pamphlet giving full directions for the movements, or, better still, to place him at first under an instructor. A regular time-table for the twenty-four hours should be made out, reserving an hour especially for the exercises; as a rule, a morning hour is best. Part of the afternoon may be spent in walking, playing croquet, driving, riding, or other out of door occupation. By such means the body may be gradually developed to a degree of physical efficiency compatible with the normal activity of the individual. Overstimulating and fatiguing exercises are, at all times, to be avoided. The same individual and discriminating attention is needed here as in the selection of protective measures.

Stimulating Hydrotherapy.—As mentioned above, applications of water at higher and lower temperatures are more stimulating than tepid water; many of the "soothing hydrotherapeutic" procedures may be advantageously used for stimulation by altering the temperature.

A cold shower bath of thirty seconds' duration is bracing, but the temperature should not be lower than 60° F. (15.6° C.). It is best given after the morning exercise, and should be followed by brisk rubbing, and, perhaps, by a short walk. Some prefer to have the bath precede the exercise, in which case it may be given in the afternoon, just before the walk. A cool sponge (60° F.—15.6° C.) with friction has a similar effect; it is most conveniently given in a tub containing very little water. Thorough rubbing with warm dry towels follows, after which the patient takes a short rest in bed. The wet "drip sheet" is also efficacious; it is given for only one or two minutes, beginning at 78° F. (25.6° C.) and gradually reducing the temperature to 60° F. (15.6° C.) or even lower.

Hot or cold douches and nozzle or jet sprays may also be employed, if given cautiously and with suitable equipment. The cold douche may be administered at a temperature as low as 50° F. (10° C.); it should last only seven or eight seconds, and should not be applied to the head and neck. After the douche the patient is removed to a warm room where active friction is applied; afterward, a short walk or mild exercise is advisable. The Scotch douche—alternate hot and cold stream—with temperatures ranging from 50° F. (10° C.) to 105° F. (40.6° C.), given with considerable force, is very stimulating. The cold application, however, should not last longer than ten or fifteen seconds.

Many of the methods employed in exclusively hydropathic institutions are, for neuropaths, too exhausting. The mistake is often made of subjecting nervous patients to too full a program of "treatments."

Stimulating Psychotherapy.—Rest, sympathy, and encouragement during the protective period have paved the way for more active and stimulating measures during the treatment by exertion. The patient should gradually be led back to an independent existence. For medical direction a courageous self-direction must be substituted. Psychotherapy in general, and stimulating means in particular, are especially suited to the management of the psychasthenic states with their doubts, fears, obsessions, and conditions of anxiety. Every effort should be made to establish self-confidence, independence, and a stoical attitude. Each individual should be studied and, when possible, induced to ride some hobby. Weir Mitchell aptly pointed out a difficulty in his "Characteristics"—the inability of the patient to "saddle, bridle, and mount his hobby." The social consciousness may be awakened, perhaps, the individual being encouraged gradually to relate himself again to others. The will must be trained gradually to the performance of the acts which, through lack of initiative, have been neglected. Tasks of gradually increasing difficulty may be assigned, until finally the self-confidence and reassurance essential to independent activity are engendered. If the patient be intellectually inclined, we may prescribe some scientific or literary work, suited to the patient's capacity; thus abstracting, the study of a foreign language, essay writing, botanizing, etc., are often helpful. Some may be ambitious to become productive workers, but they lack initiative, or do not know *how* to begin. Here is the opportunity for medical pedagogy. Some physicians have the knack of leading their patients into such work; others, it must be confessed, are unsuited for this kind of psychotherapy. Among the smaller books that may be put into the hands of patients of varying intellects may be mentioned: "Rational Living," by King; "Why Worry," by Walton; "Self Help for Nervous Women," by John K. Mitchell; "The Human Machine," by Arnold Bennett; "The Influence of the Mind on the Body," by Dubois; "How to Do It," by E. E. Hale; "Happiness," by Karl Hilty; "Cardinal Virtues," by W. D. Hyde; "Ethics of the Dust," by John Ruskin; "Courage and Youth," by Charles Wagner; "Map of Life," by W. E. H. Lecky, and "Social Rights and Duties," by Sir Leslie Stephen. Emerson's "Essays," especially his Self-reliance and Compensation, will help many toward needed independence and optimism. Even the uneducated sometimes find the little books by Annie Payson Call, "Power Through Repose," "The Freedom of Life," "Everyday Living," helpful. The more highly developed and trained cerebral cortex will enjoy Paulsen's "Ethics" (Thilly's translation) and the classics: Plato, Aristotle, Marcus Aurelius, and Epictetus. Each physician will easily add a number of other books to this list.

Return to Physical, Mental, and Social Activity.—A neurotic patient, leaving a hospital or sanatorium, or on ceasing to be supervised at home, sometimes resumes the activities of life too hastily. How often, shortly after apparent recovery, patients are compelled to turn again toward medical aid because their symptoms have come back; such patients are likely to be very discouraged, and to be disgusted with hospitals and with doctors.

To avoid relapses, proper "after care" is essential; the physician should make this clear to his patients, and it is usually well (1) to furnish a written schedule regulating the amount of work to be undertaken, and giving specific directions for the daily routine; and (2) to maintain supervision by interviews at gradually lengthening intervals until health is firmly reëstablished and the patient has learned "how to live."

Work and Occupation Cures.—For many years systematic occupational methods were confined almost entirely to the institutional care of the insane, where it was found that much of the manual and skilled labor necessary in the management of the institution could be performed by the "paroled" inmates, with considerable reduction in the cost of maintenance. General improvement in the physical and mental health of patients thus employed soon became apparent, and gradually work and occupation became adopted as valuable therapeutic agents in many of the more progressive institutions. Similar methods have also been found of service in the treatment of epileptics and the feeble-minded.

We owe the first systematic employment of these measures in the milder neurotic states to Moebius, of Leipzig, whose work-cure schedule has been widely imitated and adopted. Work and occupation cures for neurasthenic and psychasthenic states are now everywhere gaining adherents; they are doubtless destined to supplement, in an important way, the older methods of treatment by complete rest and isolation. In depressed, melancholic, hypochondriacal, psychasthenic, and some neurasthenic states systematic occupation, in conjunction with partial rest treatment, is often most useful in therapy.

Work may be mental or physical, manual or skilled, productive or non-productive. As A. S. Thayer (41), writing of work-cure, emphasizes, the work should be interesting and pleasurable; it should make sufficient demand upon the patient's attention; the patient must learn to "look out, not in." Otto Veraguth has made the following convenient divisions of occupational methods: (1) work in which muscular energy of a productive character is expended, including cabinet making, gardening, and the various mechanical arts; (2) intellectual work in art, literature, or science; (3) work expending muscular energy, but of a non-productive character, including the various outdoor sports; and (4) varied employments, including drawing, clay modeling, and wood carving.

Following Moebius's publication in 1897, occupational therapy received a strong impetus in Germany, where it has since reached a high degree of efficiency. Since 1906 definite steps toward adopting it in this country have been taken; its usefulness has become more widely recognized, and work rooms, or outdoor occupations of some sort, have been established in connection with many of the privately endowed sanitaria. The work-cure is not yet so well developed here as abroad; there is still dispute as to benefits, the indications for it, the best method of administration.

In all cases of neurasthenia and psychasthenia, as health is approached, a stage is reached in which occupation is essential. It may be desirable at the very beginning of treatment in some cases; in other instances it is in place only after rest for a period. In many of the milder cases H. J. Hall, of Massachusetts, prescribes rest for only one or two days, or perhaps a week; then, without warning, he may require the patient to perform some task. The daily program is gradually changed so that rest is diminished and work increased; in the end the full "work shop" or "occupational" course is in full swing. This author makes little use of massage, hydrotherapy, or electricity, and advocates manual work in preference to any other form of occupation; it is "objective and wholesome, and trains to accuracy and precision of movement." He places his patients in the care of a trained instructor in pottery and fabric weaving by hand, and encourages them to produce articles of sufficient merit to have a market value; if sold, the proceeds are credited to the patient's account. Willson, of Philadelphia, has had some success by combined forced feeding with work carried to the point of fatigue. He advises that the patients be fed well, but that they be required to burn up the fuel by active exercise in the open air. Most of his cases were treated, not in sanitaria, but in office practice; his patients were directed to follow a scientific schedule, in which their entire time was occupied every day for a period of six months. The mornings and evenings were spent in active work until fatigue symptoms appeared, after which rest was allowed.

Work exerts a strong psychical effect, often just as beneficial as the physical results. Mechanical labor, in general, is used for its fatiguing effects and associated metabolic changes, while skilled labor has a more distinct psychical effect by developing attention, concentration, and confidence. The psychical effect is also greatly stimulated by appealing to the social consciousness; J. J. Putnam has pointed this out in connection with the coöperative occupational methods, which have been introduced to some extent in the out-patient department of the Massachusetts General Hospital. The patients are encouraged "to meet regularly under strict supervision; to compare notes as to their success in carrying out modes of treatment that have been prescribed for them, and to gain interest, information, and enthusiasm for new efforts." The social consciousness, however, is more distinctly stimulated by institutional régime, where those who are nervously exhausted may have their new life suited to their tastes and capacities, and be taught the value of systematization and thoroughness in the performance of definitely assigned work. The progress of their coworkers, moderate competition, closer social relations, and the satisfaction arising from useful activity are some of the many advantages to be derived from associative productive occupation.

These principles have been largely adopted in Germany, where many well-equipped "work sanitaria" are to be found in which the proper applications of rest and occupation are made according to the requirements established by Moebius. All false and harmful activity is excluded; good work is done under the direction of an able and sensible instructor. These hospitals, or *"Nervenheilstätte,"* are in reality technical and academic schools, well appointed and furnishing instruction by occupation in all departments of human activity. The patients are taught how to live, and they acquire, in addition, a certain degree of proficiency in a selected trade, or in a clerical or agricultural pursuit. Colony sanitaria, somewhat similar in plan and administration to those used in the treatment of tuberculosis, have also been established for the nervous unfit. Whether or not compulsory occupation should be enforced in these institutes for neurasthenics is still a matter of dispute. Some maintain that the neurotic individual should not be driven to any task, or forced to act contrary to his natural inclination. But any serious treatment of neurasthenic states makes demands upon the patient which are not altogether to his liking. How frequently are prescriptions of rest, isolation, and hydrotherapy objected to at first, the patient stating emphatically that such treatment cannot be endured. Once accepted by the patient, however, it is the physician's duty to see that all the details of a régime are accurately carried out, regardless of the whims of the patient. In like manner, if a "work-cure" be decided upon, the character of employment being carefully selected, suited to the individual needs, it should be uncompromisingly, though tactfully, carried through.

In America some of the better hospitals and private sanitaria make some use of occupational treatment. The method is gaining in popularity; physicians are recognizing that a large part of their duty in the after-cure of neurasthenic patients consists in providing some form of stimulating and congenial occupation of the body and mind (Jacoby, 28).

Many forms of occupation may be considered. Carpentry and gardening are two types which furnish an excellent combination of mechanical and psychical factors. Carpentry, particularly, is useful because of its variety and on account of the rapidity with which productive results may be obtained, and of the interest which it stimulates. For some bookbinding, scrollsawing, pyrography, drawing, stenography, modeling, photography, or clerical work may be suggested. Grohmann characterizes basket-making and analogous occupations as "stupid." More distinctly intellectual employment may be had in art, music, literature, or science, and almost any well-chosen country place will provide opportunities for the study of ornithology, geology, or astronomy.

Supplementary Therapeutic Procedures.—TRAVEL.—If by travel were meant continuous peregrinations, it would rarely be admitted in the treatment of neurasthenia. The asthenic patient is fatigued and exasperated by the worry, excitement, and constant solicitude in the bustle and commotion of modern methods of transportation. It was formerly believed that the constant change of scenery, the absence from home and business, and the novelty of unfamiliar habits and customs incident to a prolonged voyage to a foreign country would serve to displace many of the cerebral symptoms in the overworked, wornout man of affairs; hence, a trip abroad was unhesitatingly advised. Fifty years ago, when time was less valuable and travel was slow and not so luxurious, some benefit may have been derived from an ocean voyage or an inland journey with prescribed intervals of rest. Nowadays, as Buckley emphasizes, it may be wise to choose a slow steamer, and to urge avoidance of fashionable resorts. Bouveret prefers short seacoast voyages near home to an extended tour; a few weeks' travel in the mountains in the summer, or a trip along the warmer seacoast in the winter. The "cerebral" type of patient, he thinks, fares better than the "spinal" type. Godlewski is very emphatic in his disapproval of travel for the asthenic type of patient, although he occasionally prescribes a short, carefully selected voyage for the healthy, robust, full-blooded individual with increased arterial tension. As he points out, constantly changing scenery and excitement are especially harmful to those in whom asthenopia is a prominent symptom.

Thus, while the advice to "go abroad" or to "discontinue business and make an extended tour of the country" is often harmful, still there can be no doubt that limited travel is worthy of some consideration in treatment in selected cases. We should be quite certain that the patient is physically strong enough to undertake the journey, and see to it that the itinerary is wisely arranged, especially as regards rest and quiet.

Climate.—For those whose physical condition and finances will permit them to make an extended journey the selection of a suitable health resort may be a matter of importance; if left entirely to the patient, harm often results. Keeping in mind the patient's idiosyncrasies, the principal features to be considered in selecting an appropriate resort are altitude, temperature, humidity, purity of the air, and the amount of sunshine. Repeated gray, somber, twilight days are, as a rule, depressing; while bright, sunny, moderately cool days are stimulating and exhilarating.

Some climates (e. g., seaside) have a sedative, other (e. g., mountains) a stimulating, effect. Three principal regions may be considered when we make our selection: (1) the seashore, (2) the inland sheltered resorts, and (3) the mountainous regions.

In general, as Neville Wood has emphasized, the neurasthenic should have the same climatic conditions as the aged; that is, an equable, moderate temperature in a protected locality of medium altitude. Extremes of temperature, strong winds, great humidity, and high altitude are to be avoided in all cases. The character and appointments, as well as the climatic conditions, of a resort should also be considered. The attendance and cuisine should be good, and the surrounding scenery attractive and easily accessible. The place should afford moderate entertainment and diversion, but the more fashionable resorts, where bridge, dancing, and excitement prevail, are to be avoided.

The Seacoast.—Opinion appears to be quite generally opposed to a sojourn directly along the sea border, where the climatic conditions are especially debilitating for those who are troubled with exhaustion, hypersensitiveness, depression, or insomnia. Nervous patients often fare much better a few miles inland away from the roar and tumult of the sea.

Mountain Resorts.—Allbutt asserts that a mountain residence in a dry, sunny country comes next in helpfulness to the Weir Mitchell treatment. Ziemssen, however, thought that the importance of high altitudes has been overestimated. If a bracing, exhilarating climate be desired, the more elevated areas may be sought. Too high an altitude may be distinctly deleterious; in sending a patient to the mountains he should not be permitted to go higher than 1,500 meters (4,920 feet). He should also be cautioned not to take too vigorous exercise, particularly if arteriosclerosis coexist. Eichhorst has shown that exercise in the higher altitudes aggravates most of the neurotic symptoms and is decidedly harmful. If the first week or two be spent in repose, the individual becomes, to some extent, acclimated, and may later enjoy a more active outdoor life. The advice of Godlewski may be kept in mind: "If, after fifteen days, tachycardia, insomnia, and restlessness persist, the altitude is too great or the patient is taking too active exercise." On sending a patient into the mountains it is well to advise a gradual ascent. Thus, for example, the early spring may be spent in some resort with an altitude of 500 meters (1,640 feet); later in the summer a height of 1,500 meters (4,920 feet) may do no harm.

The American and Canadian Rockies and the Appalachian ranges of the Eastern United States supply us with a liberal selection of mountain resorts. The climates of Switzerland and the Bavarian highlands offer many excellent resorts, varying in altitude from 500 to 1,800 meters (1,640 to 5,000 feet), which are suitable for neurotic patients.

BALNEOTHERAPY AND SPA TREATMENT.—The physical effects derived from the use of baths and mineral waters are so inextricably mingled with the climatic conditions of the locality in which they are given that it is difficult to estimate their value. Winternitz was probably the first to establish the fact that baths, in general, have a primary action upon the nervous system; since then numerous attempts have been made to increase the efficiency of the baths by the addition of various chemical substances. The natural waters are believed to be more efficient than the artificially prepared baths; this may depend upon radioactivity. Among the more popular baths containing chemical substances are those holding the heavy metals, salts, or gaseous substances in solution. The carbon dioxid bath has been widely used in many conditions, and has enjoyed some reputation in the treatment of neurotic states. It acts locally upon the nerve endings and the blood vessels; tingling sensations and a sense of general warmth are produced. In giving "subthermal" baths much lower temperatures may be employed if carbon dioxid be added to the bath. Bran, peat, and mud baths are also largely used. In prescribing balneological or spa treatment for a neurasthenic, a great deal more depends upon the topographical, climatic, and social conditions at the resort, and also upon the medical experience and the personality of its supervisor, than upon the chemical constituents of the waters.

DRUGS.—Pharmacological treatment of the neurotic states has varied from extreme polypharmacy to nihilism. That there is no specific for the treatment of a general neurosis is a well-established fact. The most that can be expected from the use of drugs is the alleviation of some temporary condition, or their action as tonics or sedatives. Even a hurried review of the literature upon the drug treatment of neurasthenia reveals an agreement in opinion on three points: (1) treatment by drugs has a place, but a subsidiary one; (2) that bromin or its salts or arsenic are often useful; and (3) that the use of strychnin, especially in large doses, is to be condemned. It would be confusing and of little advantage to discuss, or even tabulate, all the medical substances that have been employed in the treatment of nervous states. The fact that so many drugs have been suggested is sufficient proof of their inefficiency. Some form of drug treatment may sometimes be valuable for its psychic effect. But the psychical effect of drug treatment may be harmful if it tend to fix the patient's mind upon the condition we wish to remedy; this is especially true of external medication (ointments, plasters, blisters, setons, etc.).

Sedatives.—In cases with marked cerebral irritability a brief course of bromid therapy is sometimes helpful. The bromids may be combined with tincture of valerian or sumbul. Bouveret uses the salts of bromin for a longer period and in much larger doses than are usually prescribed. He gives from 60 to 90 grains (4 to 6 gms.) in twenty-four hours for a period of several months. Ziemssen also used them in doses of 30 or 60 grains (2 to 4 gms.) given several times daily; he especially recommended the lithium salt. For patients with decided phobias he advised that a mixture of sodium, potassium, ammonium, and lithium bromids be carried in the pocket and a dose taken whenever the "dread" appears. Dana gives bromids in 60-grain (4-gm.) doses three times daily, until the symptoms of bromism appear, and states that they may be given for longer periods if digitalis, cod-liver oil, and iron be administered at the same time.

It has been our custom to administer the bromids in smaller doses (15 grains—1.0 gm.), three times daily, and for brief periods only. They should be given after meals, and they act more quickly if given in hot milk or hot water. Some have advised the use of bromids in protracted insomnia, but usually the hypnotic action is tardy, and it occurs only after larger doses, from which secondary depressing effects are prone to follow.

Valerian and the iron, quinin, and zinc salts of valerianic acid are largely used for their sedative action. The pill of the "three valerianates" recommended by Goodell has become quite popular among general practitioners as a remedy for motor restlessness and irritability. The monobromate of camphor, zinc preparations, and cannabis indica have also enjoyed a certain reputation for their quieting effect.

Tonics.—Iron, arsenic, quinin, nux vomica, atropin, the phosphates, glycerophosphates, and simple bitters are among the drugs commonly employed for their stimulant and alterative action. Many patients appear to be benefited by brief courses of tonic treatment, especially on return to bodily activity. Dr. Weir Mitchell begins even earlier. In the third week of rest treatment he sometimes gives cod-liver oil, ½ oz. (14.7 c. c.) after each meal, and, when the full diet is resumed, 1 oz. (29.57 c. c.) of malt extract containing 5 grains (0.065 gm.) of iron pyrophosphate three times daily. If the patient be anemic, Blaud's pills or iron and strychnin in 1/30-grain doses may be given after each meal. Larger doses of strychnin, however, are not usually well borne by neurasthenics. Arsenic may be administered in any form, but is more commonly prescribed as arsenious acid in 1/100 to 1/40-grain (0.00065 to 0.0016-gm.) doses three times daily, after meals; it is believed by some to be useful in gastric and vasomotor disturbances. A course of eight hypodermic injections of sodium cacodylate (0.05 gm. at a dose) is often followed by a marked gain in body weight.

We have found the following prescription useful in the lean, underfed, anemic class of patients:

℞
Podophyllin.
Extr. nucis vomicæ
Acidi arseniosi, āā gr. 1 (0.065 gm.)
Mass ferri, gr. xxx (2.0 gm.)
Extr. gentianæ, q. s.
M. et ft. pil. No. xxx
Sig.—One after each meal.

The phosphates and glycerophosphates have been much vaunted; some prefer phosphorus in its natural form or in organic combination with the various articles of diet. In marked vasomotor relaxation ergot, ergotin, suprarenal extract, adrenalin, and sometimes strychnin have been recommended. Nitroglycerin, sodium nitrite, and erythrol tetranitrate have been used in cases with high arterial tension, but simple hygienic-dietetic treatment is, as a rule, far better. A course of injections of vasotonin (0.06 gm. per dose daily for twenty days) has been recommended in the neurasthenic states accompanying beginning atherosclerosis.

Hypnotics.—Opium and chloral are to be strongly condemned, and hypnotics in general are to be avoided if sleep can be procured by any other means. It may be necessary, however, in some cases to use drugs for a brief period in order to overcome protracted insomnia. Thus, veronal, trional, or sulphonal may be given for a few nights, and the dose gradually decreased until the drug is withdrawn. Veronal should be cautiously given, since it is not without untoward effects; alarming symptoms occasionally follow its administration even in 5-grain (0.324-gm.) doses. Paraldehyd in doses of 1 to 1½ grams (3.70 to 5.54 gr.) has been warmly recommended. Its disagreeable taste may be diminished by the addition of bitter orange syrup. In prescribing hypnotics, it should be remembered that the will power in many neurasthenics is greatly weakened and the establishment of a drug habit is to be guarded against.

Electricity.—The results obtained by the electrical treatment of neurasthenia are subject to two interpretations: Some believe that some obscure physical or metabolic change is produced, the nature of which we do not understand; others regard the benefits as chiefly psychic. If many of the properties of electricity are obscure to the medical mind, it is not strange that the lay mind should experience a strong psychic stimulus from electrical treatment. Aside from the mental effect, certain bodily changes may undoubtedly be produced. As a substitute for physical exercise faradization of the muscles may occasionally be employed to advantage. Some effect is also produced upon the cutaneous vessels; a sense of well-being and repose sometimes follows general faradism.

Sanatorium and Hospital Treatment.—Increasing prevalence of nervousness and the demand for suitable places of retreat have resulted in the establishment of numerous public and private sanatoria for the treatment of nervous and mental diseases. Unfortunately, many of these institutions are conducted in a frankly commercial spirit, without adequate medical supervision, and with too little regard for the needs of the individual patient. In some cases the medical superintendent has had little or no training in the management of nervous patients, and occupies his position largely because of his administrative ability. New systems and routine are often harmful when applied indiscriminately to all patients admitted to an institution. The intermingling of the purely functional neuroses with the insane, the alcoholic, and the drug habitués in many sanatoria is also to be deprecated.

Undoubtedly the most appropriate surroundings for the treatment of the neurasthenic and of some of the psychasthenic states are to be found in well-equipped sanatoria devoted exclusively to the purpose. Fortunately there are several institutions of this type to be found both in this country and abroad. The advantages are numerous. The general routine and system prevailing create habits of regularity in daily deportment. Irritating influences are reduced to a minimum; isolation, when indicated, may be more easily enforced; dietetic measures may be more accurately prescribed; personal control of the patient is more easily secured; and many of the more useful hydrotherapeutic measures are to be had only in well-appointed sanatoria. In addition, work and occupational cures of various sorts are more easily prescribed in specialized institutions.

In America there are good sanatoria in different parts of the country. In fact there is scarcely a city of any size which is not within convenient distance of a suitable retreat for nervous patients. In New York (Clifton Springs, Kerhonkson, Watkins' Glen), Connecticut (Cromwell Hall), New Jersey (Galen Hall), Maine (Bethel), Massachusetts (Marblehead), Maryland (Baltimore), Pennsylvania (Philadelphia), North Carolina (Pinehurst), Florida (Palm Beach), California (Santa Barbara), Colorado (Colorado Springs), Texas (San Antonio), Georgia (Atlanta), Indiana, Wisconsin, etc., are to be found excellent resorts for rest cures and the general management of nervous patients; while Massachusetts, Connecticut, and Maine in particular have several institutions in which work and occupation cures are extensively employed.

In Europe there are many well-conducted sanatoria for rest and isolation treatment. In England the physicians make much use of "nursing homes" for rest cures (*vide infra*). As examples of well-known sanatoria for the neuroses may be mentioned those in Berlin (Haus Schönow), Göttingen (Rasenmühle), and Switzerland (Kolonie Friedau).

Hospital Treatment.—The expense and inconvenience associated with the removal of a patient to a distant sanatorium, the fear of an unfamiliar institution and of a strange physician, in part account for the indecision and hesitancy shown when sanatorium treatment is suggested. If this aversion be marked, it may be necessary to choose between treatment at home and in a general hospital. Of the two the latter is, in most cases, to be preferred. The hospital should be agreeably situated, the wards and private rooms should be cheerful, and, as far as possible, free from unpleasant odors; and medical and surgical cases should be cared for in separate departments. The services of a special nurse should be engaged, and the house physician should be especially interested in nervous patients if he is to help in the supervision of the patient.

For those who cannot afford the expense of a private room or the services of a special nurse, much improvement may, in selected cases, follow a few weeks' stay in a general public ward. If the patient be hypersensitive and suffer from false pride at the thought of being in a public ward, the treatment will scarcely be of benefit. With the hearty coöperation of the patient, however, much good is often done. Rest and suitable diet can be more easily arranged for; removal from home surroundings is secured; partial isolation can be attained by means of screens. These protective measures may be easily carried out in a general hospital, but some difficulty may be encountered when the time arrives for stimulating and occupational treatment.

The present tendency in hospital treatment is to shorten the period of rest and isolation, sending the patient soon on a vacation, where he may continue a modified rest cure with selected occupational features.

We have found hospital treatment admirably suited to certain patients, and some of our results attained by simple methods are described in the American Journal of the Medical Sciences for October, 1906, p. 499.

Treatment at Home, in Nursing Homes, and in Country Places.—If we consider the etiology of the psychoneuroses it would seem that home treatment must occupy a minor place in the therapy of these conditions. In all severer cases it is contraindicated. Occasions arise, however, in which no choice is permitted, and the general practitioner is called upon to direct the management of a case in the face of conditions which would cause the most experienced neurologist to quail. Much can be done, however, even in an unfavorable environment; the results will largely depend upon the physician's supply of common sense, his personal attributes, rational sympathy, and force of character.

The patient should be put to bed in a room by herself. If a trained nurse is not to be had, some member of the family, whose wisdom, tact, and understanding are to be relied upon, should assume the duties of nurse. All other members of the family and visitors are to be excluded from the patient's room. It is often helpful to begin with a brief period of milk diet (Dubois). When full feeding is resumed general light massage may be prescribed in the morning and wet sheet pack in the evening; later on some form of diversion, reading, sewing, games, or physical exercise may be added. As far as possible, all household, domestic, and financial worries are to be excluded. If the patient be the mistress of the house, her duties should be assumed by some one of her choice in whom she has confidence. Women of hypersensitive and morbidly conscientious nature may feel that their enforced idleness is a burden to the family, or that everything must be going wrong because they have ceased to supervise the household. Their entire time is spent in wondering how affairs are being conducted during their seclusion; each day they insist that to-morrow they must resume the activities of life. Such patients do badly at home; they do better in a hospital or in the home of some friend or relative.

For those who need rest, a change of environment, or the simpler institutional methods, a well-selected nursing home may be all that is required. In England the nursing home has become well recognized as an efficient method of treating the milder neurotic states. The usefulness of this method is gradually becoming recognized in America, and there are few of our larger cities which have not one or more moderately priced homes for the care of convalescent and neurotic patients. We have not, however, fully learned the need of nursing homes devoted exclusively to the care of the neurasthenic and psychasthenic. Patients in a well-conducted home need not make great demands upon the physician; occasional visits may suffice. It is preferable, however, to have the nursing home so situated that the physician responsible for the patient may find it convenient to maintain his interest in, and general supervision of, the case.

Among the patients who are benefited by a short stay in a well-appointed country home, away from the noise and bustle of city life, may be mentioned the overworked, tired business man—the cerebrasthenic. He may quickly find relief in a quiet rural retreat, with its abundance of good food, fresh air, and repose, combined with the healthful exercise of mind and body which the activities of country life afford.

Symptomatic Treatment

To speak of the symptomatic treatment of a condition of ill health which is itself regarded as a symptomatic disorder carries with it a confession of the inadequacy of our general principles of therapy to meet the demands in all cases. No doubt in time most of the symptoms will yield to the protective measures previously outlined, and we feel that in every case it is better to give them a thorough trial before resorting to more specific symptomatic treatment. Occasionally, however, one or more features of the neurosis may so predominate as to demand special treatment directed to it alone. This is particularly true in cases where our efforts to secure rest are frustrated by persistent insomnia, pain, headache, excessive cardiac irritability, phobias, or anxious states. Fatigability, restlessness, hyperacusis, depression, constipation, anorexia, and sexual abnormalities may also demand especial attention in certain cases.

Insomnia.—As a symptom of ill health, in general, insomnia is one of the most common conditions with which the physician has to deal. Its causes, therefore, are far too numerous to permit of general discussion in this chapter, and we shall confine our remarks entirely to the insomnias associated with neurotic states.

Sleeplessness in nervous patients is of two kinds: First, there is that met with in the restless, irritable individual of "high tension" who, through an uncontrolled desire to get everything possible out of life, remains up past the normal retiring hour and finds that upon going to bed he cannot sleep. He seeks sleep, and is provoked because it eludes him despite his assumption of various postures. The entire activities of the day then begin to pass, in tormenting succession, through his mind, and the consciousness of dragging time becomes intolerable. He hears every tick of the clock; the rustle of the wind annoys him; the slightest external sound yields a perception out of all proportion to the stimulus. The second type of insomnia may be called "ill-sustained sleep," "ephemeral sleep," or "automatic wakefulness." The patient usually has no difficulty in going to sleep, but may awaken once or twice during the night, and, in some cases, at the same hour each night. In most cases he awakes in the early morning (matutinal insomnia), when the sleepless interval may vary in duration from a few minutes to several hours. In each of the two types different combinations of disturbing factors may be found requiring appropriate therapeutic adaptations. Whatever type of insomnia be complained of, the physician should first determine that it really exists. One dare not depend entirely upon the patient's statement, for patients often feel that they have scarcely slept at all, when in reality they have had many hours of sleep.

Some of the causes of insomnia in the neurasthenic should especially be kept in mind when attempting to overcome it. Pain of some kind may be the disturbing factor, and, if so, should receive primary attention. In those who have difficulty in going to sleep the cause may lie in some abnormality of the daily routine. Overindulgence in tea, coffee, tobacco; insufficient exercise; dietetic errors; worry; overwork; or the patient's attitude toward sleep—dread of going to bed because of previous sleepless nights—any of these may be the cause of persistent insomnia. The state of the arterial tension should be kept in mind; disturbing nycturia and cardiac irritability are often associated with arterial hypertension.

The relief of insomnia, then, may depend upon (1) treatment of conditions associated with the general neurosis, (2) general treatment of the nervous condition, or (3) upon specific measures directed toward the symptom itself.

As to the general treatment of insomnia, the measures employed for the general neurosis usually suffice to overcome the insomnia. The sleeping room should be in a quiet part of the dwelling; the bed clothing and general appointments of the room should be comfortable and restful. After the evening meal the patient should not be allowed to engage in exciting games, or in lively conversation, or in stimulating reading. The entire mental and physical life should be arranged in a manner most conducive to repose. As a rule, it is better to have the physician make his visit in the morning rather than in the evening, though a short visit to administer some special treatment or to offer a word of reassurance need do no harm. Frequently a brief explanation, just before bedtime, of the psychology of sleep and of the futility of "seeking" it, ending with the assurance of a restful night, is very helpful.

As to the *dietetic treatment of insomnia,* the evening meal should be light, and coffee, tea, and stimulating drinks should be prohibited. A glass of hot milk and a cracker, just before retiring, may be of service in the milder cases. Occasionally a mild alcoholic beverage, such as beer, stout, or malt, may be permitted, but alcohol is in general not to be advised. When the insomnia depends upon nycturia, no liquids should be drunk after the evening meal. If the sleeplessness, however, be independent of genitourinary disturbances, a light repast of milk or a cup of hot beef tea administered upon waking will often secure a speedy return of sleep, and, in time, relieve this type of insomnia. In some cases a glass of milk left at the bedside of the patient, to be taken upon waking, will produce the desired results.

Among the *physical agents* employed in combating insomnia, unquestionably the safest and most generally reliable application is the cold wet sheet pack. It is best given at bedtime for twenty to thirty minutes. It may be followed by very gentle massage, a glass of hot milk or beef tea, or general faradization, though, as a rule, these accessories are superfluous. Not infrequently patients will fall asleep while in the pack. In case the cold sheet is not well borne, a warm or tepid bath or pack, followed by an alcohol rub, may be tried. A wet towel around the calf of each leg is often efficacious. Some neurologists value electricity highly as a sedative and hypnotic, applying the galvanic current to the head for ten or fifteen minutes, the current flowing from the occiput to the forehead. Vibratory massage may also be of service in the milder cases.

The long list of *sedative and hypnotic drugs* at our disposal in insomnia and the certainty of temporary relief from their administration place a very harmful temptation in the path of both physician and patient.

The drug treatment of insomnia should be a last resort; one should keep constantly in mind the danger of establishing a drug habit, for the neurotic individual is prone to choose the path of least resistance. If, however, other measures have failed, or if the insomnia be so pronounced that its immediate relief is necessary, there is justification for the use of an hypnotic for a brief period. In some cases this may be all that is necessary to reëstablish normal habits of sleeping. It is usually well not to let the patient know what remedy he is taking, or the amount of it. Veronal, trional, amylene hydrate, and paraldehyd are among the most useful drugs and are less apt to give rise to drug habits. The bromids are of little service in insomnia except in the milder cases; they should be given in small doses, frequently repeated, for several days before any hypnotic effect can be expected. Morphin and chloral are simply mentioned in order that they may be condemned. Hyoscin, cannabis indica, and monobromate of camphor have been used with success where milder hypnotics have failed. If there be arterial hypotension tincture of nux vomica, valerianate of ammonia, and lecithin in conjunction with massage, electricity, and tepid baths have been suggested. In case there is arterial hypertension very satisfactory results have followed the administration of a milk and vegetable diet and the occasional use of theobromin or erythrol tetranitrate (tetrannitrol). The latter is to be given in tablets of ½ to 1 grain (0.003-0.006 gm.) each every three or four hours of the day, or during the night if the patient awakes.

Before resorting to the actual administration of drugs in insomnia one may try placing one dose of the drug within reach of the patient, telling him that if he awakes it may be taken without rising. Often the mere knowledge of having in his possession a "certain" remedy for sleeplessness will afford to the patient the assurance necessary to repose.

Headache and Psychalgias.—Pain accompanying the neuroses may vary from localized (or general) cutaneous hypersensitiveness to the most excruciating visceral or cephalgic crises. A very common symptom is a pain in the head, usually described as situated at the "base of the brain" and of a continuous boring character. Similar pains may be referred to the occiput, vertex, or frontal region. Genuine migraine, or "sick headache," occasionally occurs as an associated condition. Pain, of a paroxysmal nature, occurring in the distribution of the trigeminal nerve, is not uncommon, and may, for a time, make one suspect the existence of true tic douloureux. Rachialgia, coccygodynia, intercostal neuralgia, and mastodynia may be very distressing symptoms.

Such headaches and pains in psychoneurotics are often obstinate symptoms; they yield less easily to general treatment than do most of the other neurasthenic complaints. Massage, either general or local, manual or vibratory, gives temporary relief in most cases; occipital massage kept up for some time is often helpful. Hot and cold compresses to the head and neck, or alternate hot and cold douches to the neck, have also been of service in some cases. The full hot bath, however, not infrequently aggravates the condition and occasionally increases the insomnia from which most of these patients suffer. Electricity, either the continuous current for five or ten minutes to the head and neck, or the high-frequency current for the same length of time, is very acceptable and often seems of benefit. The Paquelin cautery may be tried. It is better not to resort to drug therapy until the simpler physical measures have been faithfully used; in case they fail a selection may be made from the long list of anodynes and analgesics available. Phenacetin, acetanilid, pyramidon, or aspirin may be prescribed in small doses for brief periods. They should not be taken by patients except under the direction and supervision of the physician, nor unless the patient is at the same time undergoing general treatment. Caffein citrate, cannabis indica, ergotin, and valerianate of ammonia have been employed with some measure of success, but their action is uncertain and at times may be distinctly harmful.

Constipation.—A frequent cause of insomnia and headaches is to be found in the obstinate constipation from which many neurotic patients suffer. "Functional" constipation may be dependent upon one or more of several conditions, among which the more important are: (1) procrastination in attending to the act of defecation; (2) atony of the gastrointestinal tract; (3) insufficient ingestion of liquids; and (4) improper diet which does not furnish sufficient residue to promote intestinal peristalsis. With these four conditions in mind certain general measures for the treatment of constipation will readily suggest themselves. The habit of attending to defecation at a definite hour each morning should be established. A glass of water should be taken upon rising, and water should be freely drunk between meals. The diet should include a generous supply of vegetables and fruits. If the intestinal atony be marked, and especially if it be associated with visceroptosis, it may be better to restrict the fluids, prescribe a dry diet, and advise the use of an abdominal binder, 14 to 16 cm. wide, to be worn constantly. The continuous use of purgatives, suppositories, or enemata is not advisable; in case it is necessary to use them for a time cascara, phenolphthalein, rhubarb, podophyllin, or aloes should be used in preference to the saline purgatives. An occasional dose of calomel, followed by a mild aperient water in the morning, is often distinctly beneficial. Abdominal massage, gymnastics of the abdominal muscles, the continuous current applied to the abdomen, and colon lavage may be useful in certain cases. (See chapter on Constipation.)

Anorexia and Vomiting.—Anorexia often disappears quickly under the general protective measures; the Dubois milk diet for a few days is especially serviceable here. It may be necessary to encourage the patient to eat even in the absence of appetite. When full feeding is resumed a general tonic or iron, arsenic, and strychnin may be given with advantage.

If vomiting be persistent cracked ice or peppermint water with a little soda may be freely given. Ortner recommends the following prescription:

℞
Mentholis, 0.5 viiss grs.
Tinct. valerianæ ammon., 10.00 iiss.
M. sol.
Sig.—Five to ten drops on sugar three times a day.

In many instances the vomiting of psychoneurotics may be controlled by psychic treatment (suggestion, reassurance, and encouragement in the effort to retain small amounts of nourishment given at frequent intervals). Atropin may be valuable in the antiperistalsis of vagotonics. Electricity may also be used for its psychic effect. If the vomiting be pronounced rectal feeding should be used for a while. It is needless to say that during treatment for this condition the patient should be in bed, and all the essential protective measures established.

Fatigability and Restlessness.—Fatigue is an early and almost constant symptom; an indication of overexertion or of diminished reserve. In either case rest is essential. Loss of sleep is a common cause of fatigue. Care should be exercised in all cases to distinguish between real and feigned fatigue; in the latter rest is contraindicated.

Restlessness, on the other hand, is frequently an indication of diminished self-control and may be either the cause or the result of fatigue. Hydrotherapy is, here, often beneficial, although, in the beginning, it may be well to prescribe the bromids or valerianates for a short period, or, in the severer cases, even hyoscin hydrobromid may be given hypodermically. The fluid extract of adonis vernalis (1 to 5 min.—0.062 to 0.30 c. c.) with bromid of soda has been warmly recommended. Since adonis resembles digitalis in its effects it may be especially beneficial where the heart muscle is weak.

Palpitatio Cordis.—Nervous palpitation should be more especially discussed under the heading of the cardiac neuroses, but a word or two may not be out of place in this chapter. Many of the cardiac irregularities of nervous origin are associated with vasomotor instability or gastro-intestinal discomfort and are often promptly relieved by treating the general neurosis. There are many cases, however, in which palpitation persists even during complete rest. The symptom is more alarming to the patient than it is dangerous; it is frequently associated with anxious states, phobias, and hypochondriacal conditions. In the milder cases it is well to ignore it, but in the severer cases, and especially where tachycardia persists, the existence of Graves' disease should be suspected. As far as possible conditions which contribute to cardiac irritability should be strictly controlled. Tea, coffee, alcohol, and tobacco are to be prohibited, and if gastric symptoms are present the diet should be light, the food given at frequent intervals, and a daily movement of the bowels secured. Such hydriatic measures as the wet-sheet pack, the tepid bath, a modified carbonated bath, or local cold applications (ice bag over heart) may be used to advantage. Psychotherapy is a valuable adjunct; drug treatment is occasionally necessary. Cardiac stimulants are to be avoided. In some cases the bromids, a mixture of the bromids, or valerian (elixir, bornyval, valichol) may be successfully employed; an occasional dose of tincture of aconite (3-5 min.; 0.2-0.3 c. c.), camphora monobromata (1-7 grs.; 0.1-0.5 gm.), or phenacetin have had good effects in some cases.

Hyperacusis.—It is customary to regard this symptom as a part of the general cerebral asthenia and hypersensitiveness, although vasomotor disturbances certainly augment it in some instances. A common complaint is "throbbing in the ears" synchronous with the heart beat; the pulsation is at times so annoying that the patient declares it to be impossible to lie upon either side. The condition is aggravated by the reclining posture or by partial occlusion of the external auditory meatus. Hyperacusis, with or without "throbbing," is often an annoying cause of insomnia. Some of these patients sleep with the head supported upon the arm or hand. This observation suggested the adoption of a very simple method which has been of benefit in some cases. The object is to furnish some support for the head and, at the same time, prevent the ear from coming in contact with the pillow. For this purpose an ear pad of absorbent cotton wrapped with gauze, and made in the shape of a circular air cushion, is placed about the auricle. It should be of sufficient width and thickness to offer comfortable support to the head and at the same time leave an air space between the external meatus and the pillow. In one instance we had an ordinary pillow so made that in its center there was a circular opening through its entire thickness, and the patient was directed to sleep with the ear over this little tunnel. By this means persistent insomnia, ascribed to continuous aural pulsations, was relieved, and eventually the auditory hypersensitiveness so improved that it ceased to be a source of discomfort and the patient was able to sleep in any normal position. In how far suggestion helped here we cannot say, but we record the observation for what it is worth. In hyperacusis all noises about the patient should, for a time, be reduced to a minimum. Later on the patient must be educated to bear sounds. It is interesting that these patients can often bear easily sounds due to their own activities (brass-pounding) while very intolerant of sounds due to the activities of other people!

Depression, Phobias, and Obsessions.—The treatment of these more distinctly psychic abnormalities is discussed under other headings.

PSYCHOTHERAPEUTIC TREATMENT

LEWELLYS F. BARKER AND TRIGANT BURROW

Psychotherapy, or psychic treatment, is, as the name indicates, the application of mental influences in the alleviation of disease. Psychotherapy may, to a certain extent, properly be applied to the treatment of all diseases; but the conditions in which this form of therapy is most efficacious, and in which it permits of extended application, belong to a class by themselves, the so-called "neuroses" in the narrower sense. Maladies with abnormal nervous and mental symptoms, but distinguished by an absence of an obvious organic lesion in the nervous system, are characterized sometimes as *"psychic,"* in that they are disorders which pertain predominantly to the sphere of the individual's mental reactions, or as "functional," in that their physical basis, if there be one, is so subtle that it is not recognizable by the methods at present at our disposal.

That the abnormal nervous and mental manifestations here considered may have neural processes corresponding to them is, of course, not denied. It is clinically convenient, however, to study, by themselves, the patient's subjective experiences as presented in morbid mental states. There may be, too, a real advantage in studying such states simply from the dynamic side, leaving out of account for the time being the question of the presence or absence of structural changes. In this sense psychic diseases may be considered to be disorders of mental adaptation, and a study of normal psychology may be expected to throw light upon abnormal mental functioning, while the careful analysis of diseasesd states will in turn give new clews for the understanding of normal mental reactions.

It is important at the outset that we bear in mind the necessary limitation of the sphere of psychotherapy to the province of individual psychology, that is, to the province of the individual's mental reaction to external stimuli.

Dealing, then, as we are, with the reactions and adaptations that represent the response to impressions from without, the factors concerned are, in the widest sense, educational. For on healthy impressions depends the healthy adaptation (or education) of a growing organism. Conversely, also, psychic disorders are often the outcome of a faulty education. For when they are the psychological outgrowth of ideas which are false, unsound, and artificial, because of inculcating a mental bias which is jarring, ill adapted, and untrue, these morbid conditions are evidence of a failure to surround the developing organism with the proper influences of education. The rational treatment, then, of many psychic disorders is essentially pedagogic, and the psychotherapeutist is, thus, largely an educator.

In a broad sense one of the first concerns of psychotherapy should be preventive. Among its primary duties is that of seeking an efficient prophylaxis against nervous disorders through a fitting propaganda of mental hygiene. Here it must provide for the suitable education of the individual from the moment of his birth, for it is in the interest of preventive psychotherapy to see to it that the proper environment be placed about the developing child. But proper psychic prophylaxis comes straightway into conflict with customs and ideas which are inveterate in the constitution of current society; how, adequately, to provide for the mental hygiene of our people becomes a sociological as well as a medical problem. It is gratifying that the National Committee for Mental Hygiene, founded in New York recently, and the various State Societies for Mental Hygiene are recognizing and facing the problem.

Unfortunately, however, it is less the preventive measures of the sociologist than the curative resources of the physician which, at present, interest the psychotherapist. Regrettably enough he is less often called upon to point out the safeguards against dangers to be avoided than to correct evils already done. Already the mind of the patient who comes to him has been imbued with unhealthy tendencies. Already an unsound, illogical point of view has been acquired by him through the long-continued ingestion of an ill-suited mental diet. In other words, the individual has received a wrong education which has to be offset by a proper one. He may, therefore, have to learn to cast off, as worthless, a whole mass of cherished conceptions, because investigation proves them to be based upon false, unsound premises; he must be taught how to supplant them with others. The process is essentially that of altering the mental attitude of the patient, acquired through the unhealthy impressions he has received, by the substitution of different impressions. It may involve an entire remodeling of the patient's mental laboratory. Such a reconstructive process, the basic principle in the psychic treatment of mental disorders, is called *reëducation.*

Of the forms of psychotherapy which may be distinguished on the ground of the particular reëducative resource on which emphasis is laid (1) that employed by Janet, of the Salpetrière, (2) that of Bernheim, of Nancy, (3) that of Dubois, of Berne, and more recently (4) that of Freud, of Vienna, are among the better known.

The distinctive feature in the therapy of both Janet and Bernheim—sometimes referred to as the Paris and Nancy schools—is the employment of hypnotic suggestion as the principal therapeutic resource. This statement applies chiefly to the treatment of hysteria as far as Janet is concerned, for Janet emphasizes the difficulty or even impossibility of hypnotizing the majority of psychasthenics. Though the Paris school sees in the phenomenon of hypnosis the evidence of a diseased psyche, to be hypnotizable is synonymous, for them, with being hysterical—and, though, for Bernheim, the condition of hypnosis appears as a normal reaction, a mere form of suggestion to which every individual is in some degree subject, the principle of treatment in the two cases is the same, viz.: To induce under hypnosis a healthy suggestion with which to offset the influence of a reaction due to the operation of morbid unconscious processes. The method of treatment in both instances is based on the idea that, when there is induced in the patient the receptive, nonresistive attitude characteristic of hypnosis, the harmful mental trend may be overcome and an appropriate impression substituted by means of beneficent countersuggestion. Thus, through hypnosis, the consciousness of the patient is put in abeyance, and access is had to the sphere of the personality which occupies a level below that of conscious perception, that is, in the psychological phrase, the psychic domain below the threshold of consciousness. This psychic realm, attainable through hypnosis, is called by Janet the "subconscious." Thus the field of operation for both Janet and Bernheim is the "subconscious."

In sharp contrast to the method of the preceding workers is that of Paul Dubois and those who agree largely with him (Déjerine, Camus, and Pagniez et al.). For the especial characteristic of Dubois's (17) principle of treatment is the repudiation of hypnosis and, along with it, of the element of unconscious suggestion as employed by Janet and Bernheim, and the substitution of a direct and rational appeal to the intelligence of the patient, with a view to explaining to him the psychic nature of his disorder and spurring him to combat the condition through a conscious effort of reason and will. It is Dubois's dictum that "nervousness" is preëminently a psychic disease, and that psychic diseases require psychic treatment. Dubois therefore uses the method of "persuasion," his treatment being primarily directed toward influencing the patient's conscious volition.

That this method is of great value in the types of psychic disorder that are amenable to reason is undeniable, and it has been much employed, before and after the publications of Dubois, by leading physicians in America. In cases thus favorably adapted the method stands vindicated upon *a priori* grounds alone. For the method is one that aims to set right a mental attitude which is biologically faulty and, perhaps, responsible for the entire disorder. It is preëminently a logical method, for it essays the correction of faulty mental habits acquired by improper training through the substitution of healthier modes of reaction. The weakened will is reinvigorated. Apathy and inertia are routed out through the stimulus of healthy incentives; the negative, reticent attitude toward life, inculcated by the morbid and enervating philosophy of the self-distrusting nervous invalid, is supplanted by the invigorating gospel of optimism and hope. So much for the theory on which the method is based; practically excellent results are sometimes obtained; but there are many cases in which success is not achieved. Nor does success in getting people well through the use of any given method necessarily prove that the theory upon which the treatment is founded is well based.

As the method of Freud, or the "psychoanalytic method," as he has called it, is one which calls for a more detailed discussion, by reason of the novelty and originality of the principles upon which it is based, we may postpone its treatment until certain preliminary topics of general psychotherapeutic bearing have been first discussed.

As to the preliminary conditions of an adequate course of phychic treatment the necessity of appropriate conditions of physical hygiene is obvious. Patients must naturally be protected against an unhygienic habit of life, such as insufficient or improper food, disturbed sleep, overfatigue, etc., as such hygienic violations militate seriously against the patient's response to psychic aids. In this connection the preceding chapter may be consulted. Suitable conditions of mental hygiene must also be provided—the avoidance of irritating and depressing surroundings, of an uncongenial atmosphere, and of contact with persons who are themselves of an unhealthy, ill-balanced mental disposition. Hence the urgency, in severe cases, of removing the patient from his habitual environment, and placing him in a different atmosphere which provides fresh associations. Thus the psychic treatment must, in every instance, take account of the psychological factors pertaining to the patient's external or environmental conditions, as well as of the internal or subjective modifications and readjustments of the patient himself. Finally, it is important to lay emphasis upon the requisite relation to the physician during the period of treatment. The physician ought to become, for the time being, the center of the patient's life. His authority should be absolute, in all severe cases superseding for the moment, by delegation, that of the most responsible of the patient's family or friends. Hence the position of the psychotherapist becomes essentially one of trust, entailing obligations that are as deep and sacred as those involved in any relation in life.

Perhaps one of the most frequent types of nervous condition is that of simple mental fatigue due to overwork and under-nutrition with incidental disturbances of digestion, headache, insomnia, depression, and the like. The cause may be obvious, consisting simply of too long and too constant mental application and unhygienic living. This character of neurosis is very frequent in students, teachers, writers, and university lecturers, and is the natural reaction to forced application, to the strain or arduous professional exactions, and to the wear of unvarying intellectual routine. The rational treatment for such a neurosis consists, manifestly, in the temporary interdiction of close mental work, and the substitution of fresh mental associations through a change of interests and environment. A sojourn in the country, a sea trip, or, best of all, perhaps, a camping expedition, with all the fresh interests necessitated by the needs of a suitable equipment for such a project, is often alone sufficient to reëstablish normal tone.

Aside from these simpler disorders the functional neuroses with psychic maladjustments may conveniently be considered for psychotherapeutic purposes under two headings: (1) Neuroses associated with certain consciously abnormal factors, and (2) neuroses associated with certain unconscious disturbing factors.

The former heading comprises the psychic tangles and disharmonies arising from causes which the patient fully recognizes and frankly avows, at least to himself, but which he may be unable, or fancy himself unable, to adjust. Psychologically the essential condition is a mental conflict, of which the logical description is worry. Worry, then, due to a psychic conflict dependent upon a disharmony among the elements of the patient's affective life may be accepted as the basic factor in the production of neuroses of this order. Where the patient's worries are warranted the treatment consists in advising and, where feasible, directing the removal of the occasions of worry. But where, on the contrary, the worries are clearly unbased, the treatment must be directed, in general, toward inculcating a robust philosophy of life and, specifically, toward increasing the patient's clearness of mental vision through logical discipline, and toward strengthening the resistive faculty through reëducative measures.

We may classify worries as positive or negative according as the conflict arises, on the one hand, from the inadequacy of the patient to the quantitative demands made upon him, or, on the other, from the inappropriateness, or quality, of the demands. In the former case the patient's responsibilities are actually too heavy. They exceed the measure for which his endowments fit him. Hence the patient labors constantly under a disquieting sense of inadequacy, added to which may be the humiliation of finding himself outstripped by his fellows. Here is afforded soil for the development of ideas of insufficiency and a train of self-deprecatory ruminations, fertile enough, perhaps, to promote the growth of the seeds of a neurosis. In such cases the primary need is an environmental change. To alter the figure, the burden must be fitted to the back; with this accomplished, that is, when the tasks set befit the patient's equipment, the situation will often resolve itself. In the latter case, that of negative worries, the patient's obligations being qualitatively unsuited to his mental powers, recourse must, in turn, be had to such external readjustments as will procure for him appropriate and congenial interests and occupations. It would be difficult to overestimate the frequency of this type of disharmony. It is often the result of an ill-chosen business, or of other faults of adaptation in the commercial sphere, and this fact emphasizes the importance of sociological factors in the determination of the psychic health of an individual—factors clearly illustrated in the undeniable hygienic asset of "success."

The psychotherapist must, then, to a certain extent assume, as an inalienable function, the task of a social worker, for he is required to take into account the sustaining influence of adequate remuneration for labors performed, and must recognize the psychological significance of pecuniary rewards in standardizing efficiency. Undoubtedly, upon the proper regulation of those sociological relations which are intimately bound up with the economic problem of wage, depends, in very great part, the psychic health of the individual, as a member of the organized social group.

The example just given will suffice to indicate that the psychotherapist has to consider, in every case, the conditions of the patient's environment, and the influence upon his psychic state of these external circumstances. The environmental conditions are summed up by Adolf Meyer (31) in what he calls the *situation*, while the response of the individual to these factors of the environment is designated by the same writer the *reaction*. Quoting him, it may be said that "it has proved to be much more satisfactory to speak in terms of situation, reaction, and final adjustment and to describe all the facts of intersection according to their weight . . ."; and again, that "the conditions which we meet in psychopathology are more or less abnormal reaction-types which we want to learn to distinguish from one another, trace to the situation or condition under which they arise, and study for their modifiability." The marshaling of the facts constitutive of the patient's life history is, then, of paramount importance. It is only through an exhaustive inquiry into the details of the patient's life, with a view to gaining possession of all the available facts, that one obtains the data necessary for an appreciation of the essential mechanisms underlying the psychic disorder—that one is enabled to understand the "situation" and the "reaction." Thus, to recapitulate, in studying the ultimate issues presented in a given neurosis the physician has to take account of (1) the individual *per se,* i. e., the "constitutional make-up," (2) the factors of the environment, that is, the "situation," and finally (3) the psychological resultant, or composite dynamic effect of these two components, that is, the "reaction." The points that are stressed by Meyer are (1) the biological bearing of abnormal mental reactions, and (2) the dynamic importance of the environmental influence in the production of psychopathological states.

This brief reference to the analytic method of Meyer forms the most natural transition to a discussion of the specific psychoanalytic method of Sigmund Freud (19). The positions of Meyer and Freud show a close resemblance, since each insists upon the study of the psychogenic influences traceable in neurotic disorders.

In approaching the method of Freud we come also to a discussion of the neuroses due to unconscious factors mentioned above. In many fundamental respects Freud's teaching marks a wide departure from the hitherto prevailing view as to the interpretation of psychoneurotic states.

On account of the widespread interest now manifest in the subject it would seem desirable to present here a brief synopsis of his doctrine. In the first place, Freud ascribes all psychoneurotic disorders to the existence, in the patient, of wishes which are unrecognized (i. e., not directly envisaged) by him. Wishes of this unacknowledged character are subsumed by Freud under the term "unconscious"; to the realm of psychic activity constituting the abode, as it were, of such unconscious trends he gives the name of "the unconscious" (*das Unbewusstsein*). Secondly, Freud regards a psychoneurosis as a spontaneous expression of the tendency toward the fulfilment of such unconscious wishes. Thus he ascribes to the neurosis a purposive significance—a moral import. The neurosis contains a motive. It embodies an underlying intention and tends to fill a void not clearly recognized and defined, because existing in the margin, outside the focus, of consciousness. Thirdly, according to Freud, the ideas or wishes which thus occupy the sphere of the unconscious possess the generic character of being, invariably, such as are ethically inadmissible by the full consciousness. Thus the psychological explanation of the creation of this limbo of the "unconscious" is to be found in the psychic conflict which arises from the opposition of consciousness to these ethically unwelcome desires, and in their enforced banishment by the tribunal of upper consciousness—a process which Freud calls the mechanism of repression (*Verdrängung,* 8). A psychic conflict, with the attendant repression of the unseemly element, is, therefore, if Freud's view is correct, the basic factor in the production of the neuroses. Fourthly, it is Freud's thesis that all such conflicts as issue in such unconscious repression have their ultimate basis in the sphere of the sexual instinct. In other words, Freud posits a sexual etiology as an essential condition of a neurosis. And, lastly, Freud's theory assumes that such symptoms as are the expression of a tendency to gratify these forbidden instincts are but an indirect, cunningly veiled representation of them, the surrogates being employed by reason of some associative affiliation with the original underlying desire. The manifestations of the neurosis are, he believes, the dramatic portrayal of the fundamental, repressed wish, subjected to a process of modification, effected by a consciousness trying to evade the real import. The symptoms of a neurosis are, in each instance, the resultant of contrary and opposed psychic trends, and represent a compromise between the two.

Such remodelings and distortions of the expressions of the brute, primitive instincts as appear in the symptoms of neurotic disorders Freud refers to as phenomena of "unconscious symbolization." It is this unconscious symbolization of instinctive trends, through which the palliation, made necessary by the censure of a conventionalized consciousness, is brought about, which is the main feature of Freud's interpretation of the psychoneuroses.

According to Freud (20) the instinct of reproduction is paramount in the life of the individual, but the sensations contributory to this impulse are originally composed of dispersed and inarticulate components, having their seats in various erogenic zones located over the body surface, and constituted chiefly of the regions of the orifices of the body (e. g., mouth, anus, urethra). The sensations arising from such erogenic zones, though present in earliest infancy, as scattered, incoherent elements, become combined and unified later, he thinks, giving rise to the characteristic sexual feelings pertaining specifically to the organs of reproduction in later life. The ultimate development of the instinct directly conducive to the biological goal of reproduction consists, then, of a process of integration which represents a product of individual evolution.

Freud teaches that there are, broadly speaking, three possible courses open for the development of the ultimate sexual life of the individual. First, the sexual life may take a normal course, leading, at puberty, to the integration of the various scattered autoerotic trends into the resultant alloerotic instinct included in the ultimate reproductive quest; there is a new sexual goal (in the male, the seminal ejaculation), having its physiological center in the organs of generation and requiring a subordination of all the erogenous zones to the primacy of the genital zone. This primacy of the genital zone, together with the finding of the heterosexual object, are indispensable transformations if the development is to result in a male individual of normal sexual life. In the female there is normally, at puberty, a repression of the erogenic zone of the clitoris with gradual transfer in normal cases to the introitus vaginæ, a feature of development which Freud holds to be of enormous importance for the origin of neurosis and especially of hysteria in the female. Secondly, there may be abnormal development either in respect of the sexual object or sexual goal due to persistence of the original autoerotic interests, and of the sexual satisfaction attaching to the primary erogenic zones, with failure of the aforesaid integration into the sexual impulse that tends toward the normal biological goal of reproduction. In this case there are presented the variations of the normal sexual impulse that constitute the different *perversions* (homosexuality; exhibitionism; Fetishism; sadism; masochism, etc.). Finally, there may be a form of development of the sexual life which is associated particularly with the psychoneuroses (hysteria; compulsory neurosis, or psychasthenia). According to Freud, the neurotics are all persons with inherited predispositions (psychosexual constitution) who have strong perverse sexual impulses, but these impulses have in the course of development become repressed and unconscious. By psychoanalysis traces of homosexuality, of failure of integration of the primary erogenic zones and partial impulses, and especially of the persistence of contradictory pairs of perverse impulses (active and passive) are discoverable. The symptoms of a psychoneurosis are, Freud believes, the expression of the sexual activity of the patient in the widest polymorphoperverse sense. The patients retain, to a certain extent, the infantile standpoint as regards sexuality, but with this difference, that the sexual impulses do not come to conscious and active expression, but are repressed and, working in the "unconscious," can manifest themselves only in the form of inhibitions; the neurosis is, as it were, the negative of the perversion.

Breuer and Freud, in their "Studien ueber Hysterie" (8), developing the earlier ideas of Breuer (1880-82) regarding the cure of hysteria by awaking, by means of hypnosis, memories of an earlier psychic trauma, together with its associated emotion—the so-called "cathartic" method—had suggested that the hysterical symptoms are the after-effects of the psychic trauma, the disagreeable emotional accompaniment of which, owing to special conditions, had been suppressed, side-tracked from the conscious psyche, and had given rise to an abnormal innervation somewhere in the body (phenomenon of "conversion"). In patients devoid of this tendency to "conversion" the idea, cut loose from its emotional element, may remain in consciousness, side-tracked from all association, while the emotional element becomes transferred to some other idea, not unbearable in itself, but made so (in an obsession) by the false attachment to it of the transferred emotion (phenomenon of "substitution"). According to this view, hysteria and psychasthenic states (compulsion neuroses) are both instances of "failure in psychic self-defence."

Freud, studying the psychoneuroses further, concluded that the psychic trauma is not, by itself, sufficient to account for the symptoms, but that contributory thereto is the memory, awakened by association, of earlier sexual experiences, usually pertaining to the period of puberty. Pushing his analysis still further back, Freud has arrived at the idea that, behind the sexual-erotic experiences at puberty, there stand infantile sexual experiences far more uniform in kind than those of puberty. Though these infantile experiences exert but little effect at the time of their occurrence, the psychic influence in later periods of life may be very much more significant. The effect is brought about, he thinks, by reminiscences of these premature sexual experiences, especially during the period of erotic imagery at the age of puberty. As the central complex of the neuroses, Freud regards the kind and degree of the psychic relations of the developing child (1) to its parents and sibs (edipus-complex; etc.), and (2) to the problems of birth and generation connected with these relatives. Psychic experiences of this sort come to all children; in order that a neurosis shall result, a quantitative excess, and an inborn tendency, are assumed, the latter being responsible for a premature and excessive development of the sexual instinct. Studies of hysterical patients have revealed, to Freud, in most cases a mental conflict between an excessive sexual repression and a preternatural need of sexual satisfaction. The outbreak of the symptoms of hysteria comes in predisposed individuals in later life as a "way out" of this conflict; the conflict is not solved, but an attempt is made to escape from it by transforming the libidinous desires into symptoms (19).

Such are some of the fundamental ideas of Freud's teaching in regard to the origin and development of the psychoneuroses. The primary factor in the production of the disorders is, he believes, the repression of the individual's sexuality, due to the ban set upon the manifestations of this elemental instinct by the strictures of social and religious convention. It is the conflict between the forces of artificial culture and those of an inherent instinct of sex, the former imposing the repudiation of sexuality, the latter insisting as resolutely upon a due recognition of the basal significance of this elemental factor in the biological economy. The sexual instinct stoutly insists that it be granted recognition in consciousness, and consciousness, in its narrow intolerance, is as fiercely resolved to debar so unseemly an intruder, to subject it to repression because it is unbearable. The situation leads to a compromise. It is agreed that the unruly element be admitted to consciousness, on condition that it soften its tone and, as it were, adopt a conventional apparel conformable to the requirements of a sophisticated consciousness. Thus it is only through dissembling that the repressed complex can succeed in evading the anathema of the conscious censor. But, though disguised, this discordant, outlawed element still lurks in the unconscious, where, acting surreptitiously, it incites dissension amid the constituents of the personality, impairing its unity and destroying the mental synthesis requisite to the purposes of concerted function. The repression, leading to the neurosis, is not wholly successful, therefore; the repressed desire waits for its opportunity to send a distorted, unrecognizable substitute, the hysterical symptom, or the psychasthenic obsession or phobia, into consciousness. A psychic repression being the essential feature of a psychoneurosis, the question for psychotherapeutics, Freud believes, is: How may disorders arising from a psychic conflict with arbitrary repression from consciousness of contraband associations be remedied? Freud, seeking to give a direct, logical answer, contends that disorders embodying psychic conflicts due to repression of elements which have a right to tenancy in consciousness are effectually to be cured only by removing the repression and frankly admitting such elements to their hereditary rights.

Since the psychoanalysis *per se* constitutes the entire method of the psychotherapy employed by Freud, simultaneously revealing the etiology and effecting a cure, an adequate account of the psychoanalytic method would entail a full description of its technique. The technique, however, of psychoanalysis has to be varied so much in different cases, and at different times in the same case, that it is scarcely possible definitely to formulate it. One of the chief technical resources, however, is the analysis of the patient's dreams; the dream is taken as a path leading into the "unconscious." The practical procedure followed in the analysis of dreams may here be broadly indicated.

The patient, having repeated his dream, is asked to relate quite freely whatever occurs to him on presenting to his mind the different elements of which the dream is composed. This Freud calls the method of "free-association." From the ultimate ideas at which the patient arrives at the end of each of the chains of associations leading from the several elements of the "manifest dream-content" the physician may be able to reconstruct the underlying trend contained in the "latent content" of the dream and so discover the patient's dream-thoughts, that is to say, "interpret" the dream. The links in the chains of associations do not succeed each other at regular temporal intervals but, frequently, the patient halts and shows signs of discomfort and unwillingness to continue. Freud assumes that in such instances the patient's flow of thought is blocked by "resistances," i. e., he has come upon a trend of thought which he has previously put away from him as distasteful, as unfit to hold a share in his consciousness. In other words, one comes at such moments upon psychic material which has been subjected to the process of repression and which, with its clusters of associations, constitutes a deterring "complex" in the patient's psychic life. Now it is precisely the release of the emotional tension belonging to these repressed reminiscences and constituting such a complex which is the central therapeutic aim of psychoanalysis.

In order to pass from the "symbols" of a dream to the "repressed element" which is symbolized, Freud has tried to develop an "art of interpretation" (21). The work of analysis is often interrupted by the "resistances" offered to the discovery of the repressed elements. The discovery and removal of these resistances are the main task of the technique. Each resistance overcome gives access to new "unconscious" material. In a sense, the cure of hysteria, according to Freud, consists in the removal of the amnesias. By filling up the gaps in memory and explaining the puzzling effects of the psychic life the continuance of the malady becomes, Freud thinks, impossible. In making the "unconscious" conscious by overcoming the "resistances" the psychoanalyst acts as an educator, one who overcomes "childhood-residues"; though the work is never complete, one can be content if he effect a "practical cure"—the "restoration of a capacity for work and love."

Next in importance to dream-analysis is the association experiment, introduced by the Wundt school, which Jung, of Zurich, has adapted to practical psychoanalytic application (29). The association experiment is useful, chiefly for the purposes of a long-continued study of some specific problem, e. g., that of differences of reaction-types and as an instrument of diagnosis. But it is also most valuable to the student in beginning psychoanalysis as a means of obtaining a preliminary survey of the general reaction of the patient and of opening the way toward gaining insight into his unconscious mental processes (discovery of resistances and painful complexes).

As to the indications and contraindications for the psychoanalytic treatment, Freud and his followers concede that the cases for which psychoanalysis is suitable are to be very carefully discriminated. It is advised especially in the "true" psychoneuroses (in the sense of Freud)—that is, the neuroses due to a psychic conflict in consequence of unconscious repressions with associative substitutions (hysteria and the compulsion neuroses with their phobias, obsessions, impulses, etc.). The anxiety-states, caused by existing irregularities of the *vita sexualis,* disorders remediable by direct removal of the exciting sexual irregularity, are not included here, nor, it would seem, are the fatigue-states, due to a depletion of the neural structure through a chemical alteration in the molecular substance of the neurones. These disease-complexes are due primarily, Freud thinks, not to psychological factors, but to organic processes caused by direct physical strain. Psychoanalysis, as a therapeutic measure, is most helpful in the psychogenic disharmonies, such as hysteria, and the compulsion neuroses, including the phobias and obsessions usually subsumed under the term "psychasthenia."

It has been objected against therapy by psychoanalysis pushed to the infantile period that the treatment is not appropriate in all cases of psychic disorders. It has been held, for example, that the association or habit-neuroses are sufficiently accounted for on the ground of their being persistent and exaggerated reactions due directly to the unconscious survival of past impressions which are of significance to the ego by reason of the strong emotional complex originally accompanying them. It is claimed by Morton Prince that such mechanisms are of themselves sufficient to account for many phobias, anxiety- and obsessional-states, without the need of invoking still other psychic incidents presumably anterior to these apparently primary affects, these latter being themselves but the reactions to former buried reminiscences. That is, it is held as unnecessary to assume the presence, in the unconscious, of those remote, infantile experiences such as the Freudian hypothesis regards as the essential etiological factors in the production of the neuroses.

One of the chief objections to psychoanalysis is made upon practical grounds. It is contended that the time required for the treatment of a psychoneurosis by the psychoanalytic method is so long that it is not often feasible and that, for the same reason, the treatment becomes too expensive to be accessible to other than well-to-do patients.

Some of the opponents of Freud's teaching are horrified at, and repelled by, the idea that sexual factors are responsible for the neuroses; even those who grant an important rôle to sexuality in certain cases deny that it is the essence of every case. Particularly obnoxious to many objectors is Freud's tendency constantly to hark back to "infantile sexuality" as the main psychogenic factor in the mental disturbance; these objectors feel that the "hidden complexes" are sometimes "talked into" the patient in order triumphantly to be dragged out again to satisfy the preconceived idea of the examiner, and they fear that harm may be done in leading the thoughts, especially of hysterical or psychasthenic girls, into sexual channels.

In reading the bibliography, one gets the impression that some of the opponents, as well as the adherents, of the views of Freud have become almost fanatical in their partisanship. We would urge that physicians, for the present, keep their minds open, and that they be on guard against being led astray or frightened by extremists on either side. It should be remembered that most physicians have been brought up in an anatomical-pathological-chemical school, and that very little attention has, hitherto, been paid to psychological studies in the medical colleges. It is well, too, to understand that the term "sexuality," as used by Freud, is much more inclusive than the coarse sensuality often meant, and that, in employing it, he refers, not to physical sexual activities only, but also to amatory imagery in the widest sense. Certain it is, that many who have given Freud's psychoanalytic method a trial in the treatment of psychoneurotic states have grown ever more enthusiastic over it. A considerable personal experience in the trial of it in the treatment of hysterical and psychasthenic patients should, it seems to us, precede any strong expression of opinion either in favor of or against it.

TREATMENT OF HYSTERIA

SMITH ELY JELLIFFE

"All that glitters is not gold." It is equally true that everything that shows hysterical phenomena is not hysteria. In any discussion of hysteria it therefore becomes of paramount importance to give some definite expression of the subject under discussion, and to separate, as far as possible, those features which tend to confuse the main issues.

This mode of approach is necessary in all fields of medicine. Modern advances in physical, biological, and chemical research have done much to sharpen our nosological concepts, thereby rendering the application of therapeutic measures understandable. In the particular field of psychiatry it may be said that the onrush of research has done as much, if not more, than in all other fields, but there are intrinsic difficulties and complexities which have not yet received a complete solution, giving the entire field an aspect of incompleteness which to the lay mind is more than confusing. In no corner of this field is this more apparent than in the domain set apart for hysteria.

Hysteria has been called the "enigma of personality." It was such for the ancients and remains one of the most actively discussed problems at the present day.

Its descriptions have entered into legends and folklore long before historical records were made. The most ancient books of the East and of the West contain in unmistakable outlines many of the phenomena precisely as we see them at the present day. The poems and plays of Homer and Euripides show its chief features as well as the writings of the modern of moderns, Ibsen. Historians at all times have had to deal with the hysterical personality. Judges and law makers have been confused by its contradictions and its inconsistencies. Physical phenomena have served as models for the adornments of ancient Egyptian temples and modern classical paintings. Priests, lawyers, philosophers, physicians, and laymen have endeavored to understand it in all ages and in all climes.

Hypothesis has replaced hypothesis; societies have been disrupted and sects rent in twain in expounding its nature, and it may safely be said that no question can create as much discussion in a modern medical society as that of hysteria.

No unanimity of opinion has ever been reached. It is doubtful if any ever will be reached. The present day Freudian advocate is subjected to abuse similar to all those who have tried to explain its fundamental features.

There are many reasons for all this. Much of the discussion would be rendered useless if people would agree as to what they are to discuss. W. James has put it well in his valuable study of Pragmatism. It all depends upon what one means by "going around the squirrel." Words are only symbols representing things—but they are fluctuating and not immutable symbols, and one readily perceives that the most active of modern disputation concerns itself with variations in the significance attached to the symbols rather than to the things themselves.

It therefore becomes imperative that a preliminary statement be made of what the viewpoint will be in the present chapter. The author who would write upon hysteria has many to choose from, even should he not have arrived at personal convictions. Even the arriving at definite and fixed ideas regarding a subject so very intricate has its dangers, for so detailed has become the analysis of personality of recent years that concepts long cherished as apparently incontrovertible have been overthrown. Thus the immutable gap that has separated hysterical amnesia from that of epileptic amnesia has been definitely bridged by the researches of Maeder, and it is no longer tenable to maintain as a proven dictum that epileptic amnesia is absolute and that this should constitute an infallible differential from hysterical amnesia.

Recognizing the great difficulty of picking out from the neurotic and psychoneurotic medley any single group with uniform features not shared by any other members of the group we purpose first to show what we shall *not* consider as hysteria, and as briefly as possible.

We shall exclude from our discussion those patients whom Déjerine

(1) has so well termed the "fausses": false gastropaths, false enteropaths, false cardiopaths, false genitopaths, etc. They are not a homogeneous group by any means, and unquestionably there are hysterics, in one sense, among them, but these are patients whose symptoms are largely the product of medical faddism. There are a large number of these purely manufactured products, not the products of suggestion, but of direct education. The less said about them the better, for of modern Molières there are plenty to criticize present-day fads in medicine.

We shall exclude the neurasthenia group. In this we recognize two fairly distinct classes: a neurasthenia proper, congenital or acquired, which is, we believe, a comparatively rare disorder; and the anxiety neurosis of Freud, which general class increasing experience serves to show to be a fairly definite group with a comparatively uniform etiological factor.

We would exclude the dementia precox group from our consideration although this is manifestly an extremely difficult task. In both hysteria and dementia precox mental mechanisms are disturbed in very similar ways and differ apparently largely in a quantitative rather than a qualitative direction. The dementia precox patient becomes much more involved in his complexes and they become incapable of discharge by adequate reactions.

Jung, in his masterly study on dementia precox (6), has drawn a striking parallelism between this disorder and hysteria, and clinical experience constantly meets with the problem of diagnosis. Thus the emotional apathy of the patient with dementia precox resembles very closely and is conditioned by similar mechanisms as the "I don't care" attitude of the hysteric. Jung speaks of the tendency for the indifference of the hysteric to suddenly blow off in a sudden wild period, a crying spell, or a burst of rage; quite similar affect discharge takes place with the schizophrenic or dementia precox patient, but it usually takes a more refined method of probing to get at the complex in the latter case. Yet when the analysis has reached the sore spot the mask of apathy of the dementia precox may be made to disappear, with quite as tumultuous an explosion, if not more so, than with the hysteric. Even the feebleminded, the idiot, and the imbecile show such explosions conditioned by the same factors. As a rule the schizophrenic guards his complexes more zealously. He shows more blocking to adequate discharge.

In hysteria, as in dementia precox, we find affective states without any adequate ideational content. There are obsessional hysterics, suffering from great anxiety, who are thoroughly aware of the groundlessness of their anxiety. They say there are reasons but they cannot give them. They are buried deep in the personality. Similar obsessive ideas are present in schizophrenic cases.

Speaking of character similarities, Jung has also called attention to the fact that one cannot in reality speak of a hysterical character. Hysteria creates no character, but it does exaggerate existing conditions. All types may be found among hystericals. Saint and sinner, rich and poor, altruist and egoist, the passionate and the frigid—all are capable of the hysterical mode of reaction. There exist within such individuals powerful complexes which are in conflict—at war—with the ego complex.

The external mannerisms, affectations of speech, of voice, of gait, originality, stupidity, are met with in the hysterical precisely as in the schizophrenic. In both it is very frequent to see the aristocratic gaits, the literary enthusiasms, the aping of the mode. In both the mechanisms are caricatures of the normal.

Regardlessness, narrowmindedness, and an inaccessibility to persuasion we find both in the normal and pathological spheres, especially when accompanied by affective causes. Under certain conditions there need only exist a firm religious or other conviction to make a person careless of others, cruel, and narrowminded (3). Most reforms and reformers show this mechanism beautifully and few followers in a reform realize that they are seeking their own selfish ends under the guise of something laudable. This same mechanism is very frequent in both hysteria and in dementia precox. It must not be forgotten, however, that whereas the resemblance is clear they are not identical, since in the schizophrenic the reaction is buried deeper and may be complicated by organic factors as yet not understood. Possibly some future worker in the complicated anatomy of the thalamus, that large station which receives the full brunt of all our sensory impressions and which elaborates the primitive feelings, may give the key to this enigma. At the present time one can only speculate.

In the intellectual sphere precisely similar anomalies are found in both disturbances. The so-called dementia of schizophrenia is more apparent than real, a fact well recognized by Kraepelin some years ago, and more recently emphasized by Bleuler. The narrowing of the field of consciousness is a very striking anomaly. Janet, in speaking of the hysteric, calls attention to the suppression of the intellectual faculties and to the difficulty in fixing the attention. Vague preoccupations fill their minds, and they cannot be interested in a work assigned to them. This is a precise description of many schizophrenics. The hysteric returns to his story, her phantasy, her fabrication—her whole being is for the time constellated about her complex. This is also true for the paranoid schizophrenic, only he is more deeply constellated. The hysterical may be dislodged from her stronghold. A therapeutic Siegfried may rescue the shut-in Brunnhilde, but as yet we know of no knight's move to get the paranoid schizophrenic out of his delusional castle.

The hysteric is not free from hallucinations, nor from delusions. Indeed, these are common materials in the building of all mental disorders. Even mental health utilizes them. The content of the hallucinations and delusions is the interesting thing. Hysteria, the A, B, C psychosis, if one may so express it, affords an excellent glimpse at more serious disturbances. Thus we may regard, with Jung, obsessive ideas as being parallel with delusions; so also may the narrowminded prejudices, so frequent in hysteria, and in many apparently healthy people; even the stubborn headaches and bodily pains of hysteria are analogous mental mechanisms.

Thus one may see how close mental health lies to mental disease, and also why narrowmindedness, prejudice, and bigotry are responsible for so much conflict in the world. They are the grit, as it were, which causes the mechanism to wear, and finally break down, and thus become unadjusted to proper functioning.

Finally, in the motor sphere one finds striking analogies between the two states, especially in the phenomena known as stereotypies. These patients become, so to speak, decerebrate automatons. Jung thinks it daring to speak of certain catatonics as such, yet they certainly are such to all intents and purposes, for a time at least. Kleist has elaborated Wernicke's ideas and has attempted anatomical explanations of these phenomena. Southard's work on the cerebellum in dementia precox supports Kleist's view, yet Isserlin's objections are well founded when he points out the incompatibility of durable lesions and transitory functional disturbances.

Without entering further into these analogies, enough has been indicated to show how closely related the schizophrenic may be to the hysteric. We are not now concerned with differential diagnosis, but are only pointing out these analogies because they may be made of therapeutic service later.

We do not purpose to deal with the group called Psychasthenia as outlined by Janet. From our viewpoint, this is a thoroughly artificial grouping of symptoms occurring in patients suffering from schizophrenia, hysteria, manic-depressive psychosis, alcoholism, hyperthyroidism, neurasthenia, and other fairly well-defined disorders. It is not, to our mind, a valid nosological group—any more than "ascites," "headache," or "epilepsy."

In other words, nothing is to be gained from the therapeutic point of view by considering psychasthenia as a nosological entity, and failure will only come from such a consideration in treatment, for it is manifestly improper to treat a patient suffering from a manic-depressive psychosis, in which phobias and obsessions are prominent, in the same manner that one would treat a beginning dementia precox having the same superficial picture of phobias and obsessions. It is particularly from the therapeutic point of view that Janet's conception of psychasthenia is, in our opinion, so unfruitful.

Manic-depressive psychoses have to be differentiated from hysteria very frequently. One of the commonest mistakes in diagnosis is the confusion of these two pictures. It is only by the proper application of the psychanalytic method that one can make a differentiation between patients whose disease pictures superficially resemble one another.

Association tests, however, reveal that the mechanisms in the two diseases are quite distinct. It is impossible in this place to enumerate the differentiation that Jung has made, and reference must be had to his valuable diagnostic association studies if one would grasp the significance of this method of analysis (4).

Finally, the various grades of feeblemindedness must be brought in close association with hysterical phenomena. So true is this that Steyerthal has gone so far as to say that practically all hysteria is nothing but neurasthenia on a feeble-minded basis. We are not prepared to accept this view, although there are many suggestive features in the statement, for certainly the hysterical reaction is a primitive type of reaction, and, in so far as its primitiveness is concerned, is to be looked upon as a feeble-minded type of reaction. Still, there is considerable difference between considering the hysterical reaction as a feeble-minded type of reaction and considering hysteria as essentially a feeblemindedness. These remarks only emphasize the significant fact that one would expect to find a hysterical reaction in people of a more feeble-minded type—indeed, the comparative studies of Kraepelin in Java demonstrate this—and thus the combination of feeblemindedness and hysteria must be considered as extremely common. It is perhaps the commonest combination that one meets in practice, and is, moreover, an extremely important feature of the situation when the subject of treatment is to be considered.

Enough has been said to indicate that the viewpoint of the present chapter is that hysteria is primarily to be considered as the expression of a particular reaction type. Those individuals show hysteria who show a certain method of meeting certain situations, which situations occur in their social environment.

These modes of reaction become habitual, and are distinguishable from normal modes of reaction solely by reason of their greater frequency, greater intensity, and greater number. This means practically that every individual has hysteria possibilities, certain individuals have hysteria probabilities, while others, again, are the real thing.

Without discussing in detail all of the various hypotheses concerning these reaction types, and the causes for the same, we feel that the most fruitful line of inquiry is that offered to us by Freud and his students. We do not mean by this that we are prepared to accept in detail all of these teachings, particularly those related by his followers, but we believe that, previous to the researches which began with Charcot, were further extended by Janet, and finally brought to practical objective demonstration by Freud, nearly all of the studies on hysteria were sterile.

It has been admitted for years, even centuries, that in all organic nature two important impulses stand out from all the rest—the impulses, instincts, or forces that make for perpetuation of the species, that is, for race propagation, and those forces that make for the protection of the individual, that is, for the struggle for existence. In comparing these two paramount forces, it is recognized that the race must continue, but the individual must die. This is the rule in all higher forms, although it has been taught that in some of the lower types, among the infusoria, for instance, there is no such thing as individual death, and that race propagation and self-preservation are coincident and coequal. However this may be, it is not true for any of the higher forms of life. It therefore becomes of the most practical importance to trace the development of the expression, and the interaction of these two great forces in human life, if one is to be able to explain the phenomena of conduct.

One might compare the forward drive of living, roughly called the libido, to two fiery animals which must be controlled. The forces of civilization are the harness by which he must be guided—convention, modesty, chastity, convenience, honesty, law—one might go on and enumerate thousands of the bits of harness that society has imposed for the control of the animal. This is a rough figure, but much human conduct and behavior can be interpreted by keeping it in mind. The tremendously complicated mutual interplay of the two forces constitutes practically all of the activities of the human mind, conscious and unconscious.

The fundamental postulate of the teachings which have appealed to many students of the present time maintains that in the psyche of the hysterical there arise certain constellations of affects, or complexes. These are primarily associated with these two large forces which determine all conduct, and their alliances with those of the libido particularly are most prominent. In the hysterical the unknown and unappreciated libido will not down. It has not found its proper forward task. Therefore, the libido, in the larger sense, introverts, and, flowing in regressive channels, takes hold of infantile phantasy. It therefore must needs have some expression, but such expression must conform to certain recognized formulæ. Being barred such expressions, often self-imposed, it therefore suffers a conversion into some form of physical or mental malady. It can then be made a subject of careful attention and solicitous apprehensive care on the part of those about the hysterical individual, and in the converted hysterical symptom, be it paralysis, or blindness, or deafness, or any of the thousand and one various physical symptoms possible, the individual seeks and obtains his relief.

This is but a very hasty sketch of the mechanism of conversion. It has been made short because the forms of mental mechanisms have already been discussed in this book in the chapter on Psychotherapy. In this chapter one may find the main outlines of the modes and methods to be pursued in the analysis of these hysterical patients (also 5).

Inasmuch as the treatment of an individual suffering from a form of disturbance which we call hysteria must take the personality of the individual subject largely into account, it is not to be wondered at that no two individuals will be in full accord in all of the details of a particular case. Therefore, one finds that one author accentuates one feature in psychotherapy, and another another. This is due largely to the fact that they may be dealing with different individuals. It is quite conceivable that one student should lay stress upon the value of hypnotism, in contrast to the value of psychanalysis, if the former has in mind that mixture of hysteria and feeblemindedness which is incapable of reacting to the intricacies of the psychanalytic method. Such persons are most effectively reached by hocus pocus, be it in its refined scientific form, hypnotism, or in a much more crude and crass form of quackish methods.

For the true, outspoken hysteria, with evident conversion symptoms, we feel that the best mode of approach is the psychanalytic method. This does not mean that all such patients will require months and months of treatment by any manner of means. Many of such patients apprehend the idea of the mechanism in a few hours. They are anxious to get well, they grasp in a moment the tricks that the nervous system is playing upon them, and they are enabled to carry out their analysis and obtain relief in a very short time. Others, however, in whom the relief-seeking conversions have been established for years, and in whom habitual reactions have become a part of their very being, often require many months of careful analysis, and of coincident careful reëducation.

Hypnotism, psychanalysis, reëducation, these are the usual weapons used. The end and aim of all is reëducation. The most capable of the tools, not for all cases, but for the most difficult, is psychanalysis. Without it a comprehensive rehabilitation of the attitude of the individual toward life, toward himself, and his difficulties we feel can never be gained. It is recognized that for many patients such a mode of procedure is time thrown away. We might compare the use of psychanalysis to the use of cathartics. There are many patients for whom a single cathartic relieves a disturbing constipation. There are others who require months of treatment to overcome a difficulty in the bowel function, and it is just as absurd to assume that the single dose of cathartic will heal a chronic condition as it would be to start a long complicated dietetic régime upon the patient, for whom a grain of calomel would settle the whole difficulty. So in the problem of hysteria it is equally true that for some a word of common sense, a strong insistent personal appeal, especially in the beginning, will entirely overcome and reëducate a starting hysteria reaction, whereas for others it requires months and months of careful, patient treatment. The practical therapeutic difficulty is in being able to recognize the extremes, and the various gradations in the means, and to apply the appropriate gradations in treatment.

Psychanalysis has given a sharp-edged tool to get at a certain number of extreme cases, which heretofore had been the most difficult in the domain of medicine. It moreover provides a most fascinating tool for the physician, one that enables him to get into the personality of the patient in a manner hitherto unappreciated, save, perhaps, by the poet, who, by means of his peculiar composition, has always possessed such an instrument.

One word may be said regarding physical adjuncts to the methods already outlined. It is self-evident that if, in the course of the treatment of a hysteric, difficulties of a purely physical nature arise, such should be corrected. It is highly important that no undue emphasis should be laid upon such disturbances, because they frequently provide a fixation point, as it were, for the converted mechanisms to settle upon. It is for this reason, furthermore, that extensive electricotherapeutic and hydrotherapeutic measures and other forms of therapy should be used with a clear comprehension of what one has in mind. It is not honest nor fair to the patient to make him rely on physical modes of therapy as the real healing methods. They are accessory. They are helpful. They are useful for improving the physical tone. They are highly desirable in many cases in relieving the tedium of an otherwise unoccupied day. They provide change in direction of thought and interest. They should also be utilized by the physician as opportunities for reëducation. The patient should be told exactly what the action of the physical agents really is, that they can go a certain distance but no farther, they are contributory to the cure, but that real adjustment must come from the mental side, from the readjustment of the individual to the eternal verities of life.

TREATMENT OF THE MIGRAINES

Smith Ely Jelliffe

Three general groups of migraine headaches may be distinguished: Ophthalmic migraine, the more usual and classical type; ophthalmoplegic migraine, which is the usual type, complicated by signs of ocular palsies of various degrees; and symptomatic migraine, associated with organic disease of the brain. The last has been already partly considered in the section on intracranial growth headaches; ophthalmoplegic migraine calls for the same treatment as a migraine plus a possible organic cause (syphilis, tumor, etc.).

Ophthalmic Migraine

(Hemicrania, Sick Headache)

This general type of headache has been known and described for centuries. Aretæus, Celsus, and Galen wrote of it. Galen apparently gave the name hemicrania from the frequent unilaterality of the disorder. Tissot in 1784 wrote a monograph which was authoritative for ninety years, to be superseded in 1873 by that of Liveing on "Megrim," which still remains an invaluable classic.

A well-marked attack of ophthalmic migraine is classical and stereotyped. The earliest descriptions leave little doubt as to the nature of the headache. The patient first notices peculiar eye sensations, a faint blur appears as one reads or looks at an object, partly obscuring the vision. Then chilliness supervenes and the blurring becomes more marked, and peculiar zigzag scotomata (fortification scotomata, often colored) appear. The chilliness becomes more manifest, and then a soreness comes on one side of the head, often behind the eyeballs, and gradually pervades one side of the head, becoming more and more intense. The scotomata disappear after from 15 to 30 minutes. Nausea and vomiting then develop, the headache has become splitting, increases on the slightest exertion, bending over becomes impossible, and, finally, after persisting for from a few hours to a few days, the patient is perfectly well.

While this bare recital of events is sufficiently characteristic to afford a diagnosis for the clearcut types, the symptomatology of ophthalmic migraine is much richer and infinitely varied. There are very few individuals who at some time in their lives have not had an attack of ophthalmic migraine, and a large proportion have had one or more of the classical type here outlined, but the full-blown attack is also comparatively rare in comparison with the abortive or variant migrainous attacks.

It is very significant that sufferers from migraine are keenly alive to the variants of their attacks, and it becomes necessary to outline some of these variations if a proper diagnosis is to be established and an adequate therapy followed.

Abortive Attacks.—These are common with the migrainous individual. In some sufferers the abortive attack usually begins in the classical manner, with chilliness, partial scotomata, and depression. The patient is apprehensive and waits for the coming ordeal—often lies down, and finds that, after a half hour or more, the attack passes off, leaving only slight wretchedness or a dull, heavy head with sore eyeballs.

Again, the attacks may consist of the scotomata alone without the vasomotor spasm and chilliness or headache. The depression alone may be severe enough with the other symptoms just marked to give a clew as to the cause of the mental state.

Variants or Equivalents.—Even more common than the abortive attacks are the variants. The most frequent is that unaccompanied by marked prodromal symptoms. The patient develops a severe hemicrania or a bilateral headache with sore eyeballs, and may or may not have the nausea and vomiting. If the life history of a series of headaches in a migrainous individual be followed, it will be noted that scotomatous attacks vary considerably. Some patients will run a series of migraine attacks, always accompanied by scotomata; then a series without any scotomata at all. Sometimes the attacks with scotomata occur only at night, and the patient awakens with a headache. Possibly he may have dreamed of seeing fire, which is the only incident that points to his having had scotomata.

Many individuals skip the nausea and vomiting in the majority of their attacks. One patient with a life history of over 200 recorded attacks had nausea in about a dozen, and vomiting in only one. Yet the other features of the headache were classical.

It is impossible to state percentages of prodromal chilliness or scotomata, unilateral or bilateral involvement, length of time of pain, nausea or vomiting, because each individual's migraine history varies and each attack varies.

With the majority of the migrainous the severity and number of the attacks usually diminish regularly with advancing years; again, in a smaller number, the reverse situation is met with.

Among the rarer migraine equivalents are isolated attacks of nausea and vomiting, isolated paresthesia, attacks of giddiness or vertigo, intestinal and bladder disturbances, hysterical outbursts, etc.

The short history of one patient who kept notes on 168 attacks will perhaps show some of these facts in another light (1). These attacks were observed in ten years. More than 75 per cent. of them occurred in the first three years. Of these attacks 100 were abortive: chilliness, scotomata, with dulness in the head, but no marked headache. Of the 68 full attacks, one-half were unilateral, the other half bilateral, without scotomata; the rest (34) with marked scotomata and blindness. Throughout the whole series there had been no vomiting; nausea had been occasionally present with anorexia. Two of the attacks were associated with paraphasia, fifteen with sensory tactile phenomena, pins and needles in the fingers, hyperesthesia in lower limbs, chiefly at night, in six. Blepharospasm was extremely frequent during the attacks.

Another patient had a series of daily attacks for eight months, extending over a period of two years. The attacks were always preceded by chilliness, heaviness, scotomata, and blindness, lasting almost exactly 15 minutes in each attack. After ten minutes the scotomata would slowly recede, and the headache gradually mount. The greater number of attacks were bilateral. Unilateral attacks were more severe, and accompanied by nausea and anorexia. The bilateral attacks gave a dull, heavy, stupid head, but not the severe pain of the unilateral attacks.

Thus an idea may be gained of the marked variability in the attacks in different individuals, and even in the same individual at various periods.

Therapy.—In the first place, it is essential to separate the specific treatment of the attack from that of the general treatment of the migrainous constitution which renders attacks more liable to occur.

In the treatment of the attack the methods at our disposal are fairly satisfactory. It should be remembered that practically every well-developed attack of migraine shows at least two phases: a primary phase of vasoconstriction, followed by a secondary phase of modified vasodilatation. Therefore all remedies utilized for the treatment of an attack must be chosen with the stage in view.

Thus, it is futile to utilize nitroglycerin when the stage of vascular dilatation has already taken place, and vasocontractors are disastrous in the beginning stages, when the spasm of the vessels is the most prominent part of the picture.

If the patient is seen early enough in the attack, and symptoms of vasoconstriction, such as pale, cold extremities, chilliness, general misery, are present, vasodilators and analgesics should be used. Such vasodilators, however, should be chosen among those whose action is self-limited and more or less fugacious. Nitroglycerin has been found to be one of the most valuable of these, but, as has been remarked, it is only of service in the very early stages, and very frequently is then inefficacious. Of the various analgesics, the anilin derivatives are the most powerful. Acetanilid, antipyrin, pyramidon, and its allies, in doses of from 5 to 10 grains (0.3 to 0.6 gm.), are useful. Phenacetin and its congeners are less powerful, but in certain respects better adapted to universal use. Occasionally salicylic derivatives and other members of the group are of special service.

It is important in the choice of an analgesic to remember whether its action is complicated by an effect upon the blood; furthermore, the solubility, time necessary for absorption, variations in the chemical formula, the duration of action, and the character of the after-effects; these should all be kept in mind. While one is apt to regard the whole series as acting in the same way, closer scrutiny will show distinct variations along the lines just indicated. These variations may be utilized to advantage in each case.

It should not be forgotten, furthermore, that a remedy which has proved ideal, either alone or in combination, for a certain number of attacks may soon lose its efficiency. This is generally due to the establishment of a tolerance which prevents this particular combination from being further valuable. One must, therefore, vary the drugs used and also the combinations.

In general, a mixture of two or more analgesics is more efficient than a single one. In the mixture smaller individual doses can be utilized, and a certain amount of therapeutic play can be introduced, which single dosage does not permit. Thus, a mixture containing antipyrin and phenacetin gives the rapid solubility of the one, and its quick action, with the retarded solubility of the other, and its more prolonged action. A small dose of acetanilid can be combined with a larger dose of phenacetin, minimizing the cardiac depressing effects of the former, and at the same time utilizing to the full its powerful analgesic properties, a continuance of which is carried on by the latter. In fact, by a judicious combination of the analgesics with small doses of the bromids or small doses of vasodilators, one usually obtains very prompt and efficient action in the treatment of the early stages of a migrainous headache. So effectual may a carefully thought out combination prove that a headache which has heretofore been regarded with terror ceases to cause any particular apprehension in the mind of the sufferer. This is of great service in avoiding the use of the opium derivatives, which in previous times was a very potent factor in the production and perpetuation of this drug habit.

The therapeutic indications in the later stages of the attack vary only in so far as the physical signs relative to the cardiovascular system are different. If flushing, intense throbbing, and injected conjunctivæ indicate a vascular hypertension, vasodilators are of little or no value. Caffein is very frequently combined in such cases with the analgesics already mentioned with a certain amount of advantage, but, as a rule, it is not a sufficiently powerful remedy or not prolonged enough in its action to be relied upon. It is very useful in abortive and mild attacks, and particularly valuable in certain toxic forms of migraine, especially those due to alcohol, tobacco, or opium and its allies. Bromids and chloral may be combined with the analgesics, the former particularly for those individuals in whom there is considerable motor excitement, and those in whom hysterical, agitated, emotional reactions are prominent. In a few instances it may be desirable to add codein to the prescription, or even minute doses of morphin. This is particularly true in certain individuals in whom the danger of contracting the morphin habit is either not probable or under circumstances where the contraction of such a habit would not be so reprehensible, as in certain very old individuals. [When morphin is necessary, it is wiser to give it, never to prescribe it.—Editor.]

In previous times aconite, belladonna, cannabis indica, gelsemium, and drugs of related nature were widely employed, but, with the advent of the analgesics already mentioned, the use of these earlier remedies has been shown to be comparatively unimportant. Only now and then does one find it necessary to recur to these much more powerful narcotic drugs. There are individuals, however, who react much better to small doses of aconite or cannabis indica than they do to any of the analgesics, and one should always keep them in the mental eye in the treatment of the more persistent and frequent migrainous attacks.

If it is possible a brisk saline laxative should be administered, and the patient should be undressed and lie down in a darkened room, well covered up and kept warm, after having had a hot bath. Cold, as a rule, is distasteful, although occasionally ice applied to the head has been found to give relief.

A frequent error in the treatment of attacks of migraine is failure to vary the therapeutic procedure. This usually brings about tolerance and defeat. The abortive attacks, violent attacks, and classic attacks not only need different modes of approach, but a different general course of procedure is desirable during an attack.

Treatment of Constitution.—In discussing this feature in another place (1) I have said that, "although the treatment of the acute attack is fairly satisfactory, it cannot be said that we are as yet in a position to completely prevent attacks. The treatment of the habit, or the constitution, or the liability, or whatever it may be called, is a difficult problem. There is very little unanimity of opinion among students as to the best mode of approach.

"If it be assumed that the hypothesis already outlined is the most valuable one for the purposes of therapeutics, it is essential to search out all those peripheral causes which may be factors in upsetting the balance of the vascular control held by the sympathetic nervous system. A great many migraines have been completely wiped out by the correction of minor peripheral anomalies. Just what proportion of such are cured by special measures is difficult to estimate, as specialists in their respective fields usually claim 100 per cent. of cures by this or that procedure, a manifestly illogical attitude. It is certain, however, that a small proportion of patients are cured of migraine by relief from eye strain, from diseased turbinates, from habitual constipation, from adenoids, from dysmenorrhea, and following relief from a number of other minor yet definite peripheral defects.

"Whether such migraines belong to the category of the severer migraines it is difficult to say. On the other hand, one finds that certain migraines resist every form of therapeutic attack, and, in spite of the fact of many years of correction of these minor defects of organization, which are almost universal, they persist. Personal experience indicates that this is the rule rather than the exception, but at the same time it is folly to proceed on the general assumption that the correction of these minor defects is unimportant.

"Stated very briefly, the viewpoint that assumes that no migraines are due to the minor peripheral ailments is as absurd as to claim that all migraines are due to any particular form of such defect, such as eye strain, constipation, autointoxication, gastroptosis, or other defect, as faddists so frequently claim.

"Inasmuch as the attention of mankind is more or less chronically riveted upon its stomach, it is not unnatural to find this factor loom large in the history of migraine. Nearly all sufferers from it will complain that they are bilious, in which word one recognizes the fad of a previous generation, a direct descendant of the days when black bile was considered of so much importance in medicine. If the purely vegetative character of the gastrointestinal tract be kept in mind, it would seem that only grave disturbances would be provocative of such a constantly recurring type of phenomenon as migraine. At the same time, enough experience seems to have been accumulated to demonstrate, in the minds of the sufferers at least, that they are not entitled to certain gastric indulgences without the recurrence of a migrainous attack.

"Under all circumstances, therefore, it would seem desirable that a fairly commonsense gastrointestinal hygiene should be carried out. Such a hygiene should not go to the extremes of dietary faddism, but should be founded upon a common sense recognition of the individual's likes and dislikes and capacities. Certain empirical facts are entitled to considerable recognition, for it is well known that in some individuals carbohydrate intake almost invariably produces a migraine, while in others large quantities of fat provoke a like reaction. Again, in others the use of certain alcoholic drinks induces the same type of reaction.

"This is not the place to prescribe just what measures should be followed out in the correction of the various minor defects which may have some relation to migraine. The point of view of the physician should be that of the inquirer rather than that of the maker of dogmatic assertions regarding these factors. Many false gastropaths are manufactured by the physician in his attempts to carry out a régime of gastrointestinal hygiene for the relief of a recurring migraine. This suggestive factor is to be avoided, because the results are often worse than the disease.

"If the varying elements mentioned have any real relationship to migraine, it is evident why such a variety of measures will be of help to a few, and why so many more will be worthless for many. Medication between attacks is largely useless, save, naturally, in the symptomatic migraines. General medication, for no definite purpose, but just in hopes that it may do some good, as the giving of iodids, bromids, strychnin, etc., is senseless. If definite factors be found that need correction, and can be so modified by drugs in the required direction, then they will prove useful. Thus iodids will undoubtedly help many presenile arteriosclerotic migraines; bromids are useful for irritable and sleepless conditions which provide a good foundation for the nervous instability that permits an attack; laxatives are called for if persistent constipation bears any causal relationship.

"Complicated systems of diet have been devised. Usually such are more prolific in engendering semi-invalidism than useful for migraine. Here and there a patient derives benefit from a strict dietary régime, but, unless there are real reasons why a patient should not eat red meat, or tomatoes, or other sundry articles, as determined by actual experience, and under repeated experimental trials, in order to eliminate faddists' errors, the patient is better off without a diet card. The reasons sought for are not those contained in many treatises on dietetics, in which medieval notions concerning differences in red meat and white meat, vegetables growing under the ground and those above ground, are foolishly perpetuated. The only satisfactory manner to attack the metabolic problem is to carry out a complete metabolism analysis. Haphazard attacks here and there lead only to premature and insecure judgments.

"Complete formulas for attacking excessive bacterial putrefaction are applicable only when it is proved that such excessive bacterial action exists and has a relation to the migraine. The hypothesis cannot be excluded *ex cathedra,* but it remains unproved for most cases, and of doubtful applicability in a few.

"The avoidance of alcohol and tobacco, while advisable, is so only relatively. The individual's reaction to all influences should be rigidly estimated before those usually self-evident restrictions are imposed in the name of health.

"In certain individuals a change of occupation may be absolutely necessary, but here again one must be wisely conservative, and not consign all migraine patients to an outdoor life, especially when outdoor workers are by no means exempt and ought to be clerks. The character of the work is to be borne in mind. The elements of haste, of pressure, and of lack of leisure are to be thought of in this connection."

TREATMENT OF HEADACHES

The struggle against headache, considering it as a disease, or as a symptom, has been waged for many years. Cullen, writing in the 18th century, remarks that "Headache as a disease is obscure, as a symptom difficult. It may be allowed to be generally symptomatic; but I presume it may also be primary; and much confusion has arisen in the attempts to distinguish between them." He then launches into an attack on the system of Sauvages, that doughty nosologist of the 18th century, whose species were a veritable Garden of Allah.

It may not be without value to enter somewhat into Cullen's spirit and see what primary and what secondary or symptomatic headaches were recognized at a time during which American medicine was first being fashioned, and for the most part at Edinburgh, by this great teacher.

Cullen rejected from Sauvages' categories: (1) All those pains depending upon typical affections of the external parts, which may occur in other parts of the body, and the seat of which in the head changes neither their nature nor their indications. Thus, the cephalæa syphilitica is not a disease different from a pain in the shins, from the same cause.

Upon the same grounds he rejects cephalæa ab acrimonia, hemicrania ocularis, odontalgica, sinus, purulenta, ab insectis.

(2) All those that are manifestly symptomatic, such as the cephalalgia catamenialis, hæmorrhoidalis, stomachica, febrilis, pulsatilis, intermittens, gravidarum, inflammatoria, catarrhalis, cephalæa arthritica, febricosa, polonica, and hemicrania coryzæ, hæmorrhoidalis, nephralgica, and, he adds, I think on the same ground the cephalalgia hysterica, melancholia, hemicrania clavus, and lunatica.

(3) The cephalalgia anametropa. The whole species of Sauvages are thus rejected, except three, cephalalgia plethorica, cephalæa serosa, and cephalalgia metallica.

This enumeration looks strangely familiar when one glances over a work dealing with headaches, even of the present day.

But, when Cullen leaves criticism to take up description, his reliance upon temperaments, upon the phases of the moon, and the influence of humors causes us to turn to present conceptions, with some misgivings as to how long they, too, will stand the test of further experience.

Sauvages distinguished cephalalgias, cephaleas, and hemicranias—just such a tripartite arrangement may be justified at the present time. It would consider those head pains that are about the head, from the teeth, sinuses, sore muscles, etc.; those pains within the cranial cavity proper, affecting cerebral, or extracerebral, substances, and those pains which may be classed as migraine, and which for practical purposes it is just as useful to consider as a separate entity, as it has been ever since Aretæus first tried to isolate it from the other headaches, and since Galen gave it its name.

Such a division has value only from the standpoint of convenience, and will be followed here.

Headaches of Extracerebral Nature

Here are to be found a number of affections, with persistent and uncomfortable pains in the head. They are either neuralgias, myalgias, or occupation neuroses (muscular), traumata.

Neuralgias.—The ordinary trigeminus neuralgia, tic douloureux, is not to be considered as a headache; yet frequently, when the supraorbital branch alone is involved, the diagnosis from other types of headache, particularly migraine, brain tumor, pachymeningitis, hysteria, neurasthenia, eye muscle neuroses may arise. Here one finds the topographical localization sharply marked, when tested by the proper esthesiometers. This localization is either frontal, extending to top of cranium; unilateral, as a rule, the base of the nose, the upper eyelid, the ethmoid, deep within the upper nasal region. There is a characteristic pressure along the nerve trunks, and the tender points at the supraorbital foramen, and ethmoid point, the parietal tubercle, and the inner angle of the eye. There is also frequently increased sensibility to cold when tested by a cold key or other cool object.

Not infrequently an antecedent influenza, malaria, typhus, or other infectious disorder is the exciting cause. Constipation has been known to occasion such a trigeminal neuralgia; also intestinal parasites. Leukemia and diabetes are of moment.

Affections of the middle and inferior branches are omitted here, although it may be pointed out that carious teeth at times cause a chronic temporal headache, often getting worse at night and preventing sleep, whose cause may be unsuspected for some time.

Occipital Neuralgias.—These, when occurring in the branches of the occipitalis major, minor, or auricularis magnus (Hasse), often are to be separated from occipital intracerebral affections, caries of the vertebræ, cervical cord tumors, and from the frequent so-called neurasthenic headache.

These neuralgias are almost invariably bilateral, and the Valleix points may be found along the cervical outlets. When sharp in character, their neuralgic nature is obvious, but they are frequently dull, are increased, or brought about by movements of the head. The pains often reach the vertex, and are significant of this neuralgic type when coursing along the shoulders, or down into the arms.

The therapy is by local application of heat, local rubefacients, mustard plasters, cantharides, menthol, etc., special measures.

Naturally foreign bodies, sclerotic areas, wound scar tissues, etc., should be removed when they press upon the nerve terminals. Occasionally one has to deal with a syphilitic neuritis of these nerves. The Wassermann reaction should be utilized in making a diagnosis. Malarial cases need quinin. Such cases have regularly recurring pains, and the drug should be given in doses of from 15-20 gr. five to six hours before the expected attack. Iron salts, calcium, and arsenic are indicated in the anemic neuralgias.

Electricity has special indications in these more obstinate neuralgic headaches, although less frequently needed in the supraorbital type than in true tic douloureux. Here the rapidly alternating current of Leduc is of value in 5 to 10-minute sittings, once or twice a week. In the milder cases the galvanic stream is helpful. In either case the anode is placed over the sensitive pressure point, and a stream of not over 1 to 1.5 milliampères allowed to pass for from 10 to 15 minutes. Faradic penciling of the tender skin areas for from 5 to 10 minutes should then follow.

The local application of methyl chlorids will cure some of these supraorbital neuralgias.

Of the antineuralgics the antipyrin, phenetidin, and salicyl derivatives are the most useful. Antipyrin, salipyrin, aspirin, phenacetin, acetanilid, pyramidon—these are among the most useful, singly or in combination, according to other etiological conditions. Acetanilid is to be administered never in doses over 5 gr. (0.3 gm.) as a first dose to an unknown adult. Although all of these derivatives are closely related pharmacologically, as well as chemically, it will be found that individual idiosyncrasies exist that make it important to try one after another in order to find the most valuable in a minimum dose, either singly or in combination. Frequent changes are advisable, and the physician is lax in his obligations to his calling if a drug habitué results.

The use of gelsemium, gelsemin, aconite, aconitin, atropin, cannabis may at times be required, but only as surrogates to other remedial measures.

The question of massage is difficult to pronounce upon. One will frequently find patients who have found relief from deep massage, special massage, osteopathic massage, etc., after the family physician has failed. It is highly probable that such cures are the result of encouragement, of reëducation, of suggestion, and some humbug; not an uncommon mixture in all prescriptions. But one does not refuse to use a palatable vehicle to properly carry a combination of efficient remedies.

The treatment of the more frank neuralgias by injection of alcohol and by surgical procedures is discussed elsewhere.

Cervical Sympathetic Headaches.—These may be neuralgic in nature, but more frequently alter the statics of blood pressure, and more properly belong to the intracranial causes.

Reflex Tenderness of the Scalp.—Empirically it has been recognized for years that certain visceral disorders are frequently—almost invariably—associated with reflex pains or tenderness in the scalp. These reflex pains are very often complained of as headache. Hilton, Dana, McKenzie, and particularly Head have studied these reflex disturbances, and have shown, for many at least, that these skin areas are in anatomical relation, through collaterals in the cerebrospinal axis, with the main nervous trunks, coming from definite organs. When these organs are diseased or functionally disturbed, reflex pains appear in the scalp areas referred to.

The intensity of the hyperesthesia in these areas varies widely, and usually bears some proportional ratio to the intensity of the visceral disturbance.

On examining the skin areas of the head with the rounded glass head of a sharp pin, with the point, with von Frey's hairs, or other esthesiometer, one can map out these areas, which do not in general conform to any strict nerve topography. They are usually circumscribed and bilateral.

The sensations complained of are dull aches, casque-like bands, sore spots, very tender areas to touch, with persistent dull and annoying ache.

The accompanying figures illustrate the main features observed.

The therapy is twofold. Local application of counterirritation helps the tender spots, as well as the visceral disorders; adequate treatment of the visceral disorder relieves the topalgias, or localized pains.

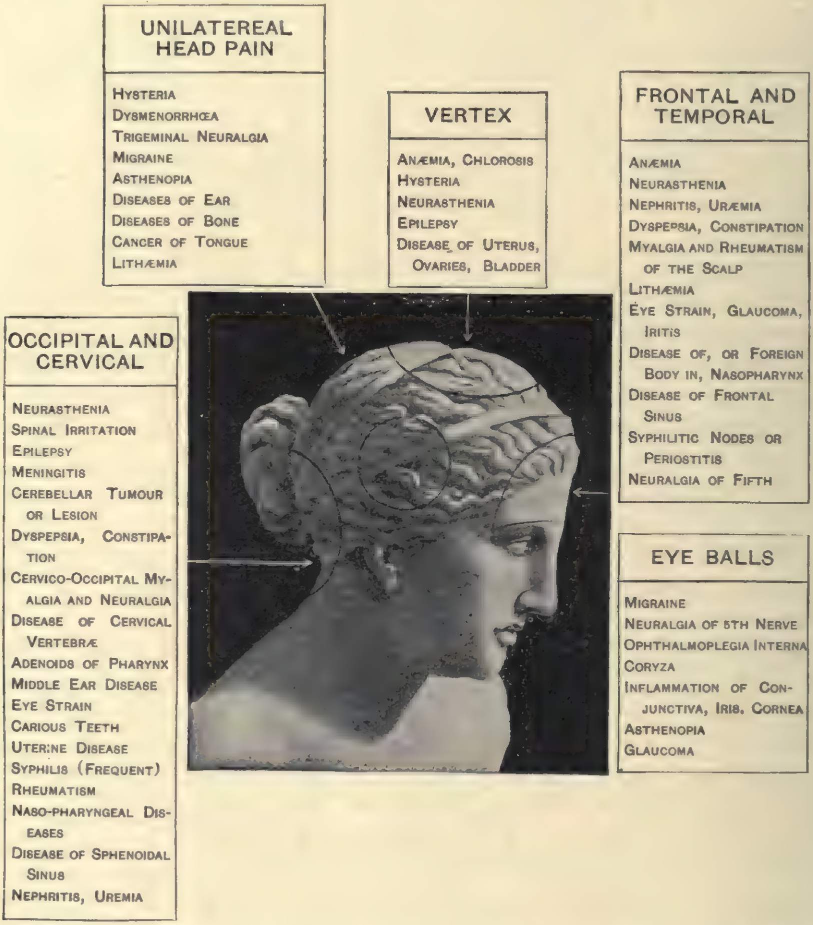

Fig. 1.—The General Diagnostic Indications to Be Derived from the Seat of Pain in the Head and Face. (Butler.)

Neurotic Muscle Headaches.—This group of local headaches is very important and little understood. I purpose to include here a number of headaches which really are the result of a continuous muscle activity. This is not a conscious activity in the sense of a continuous series of muscular acts, but is rather the result of a series of partly automatic motor adjustments, usually set in operation by reason of some anomaly in the chief receptors of the head region, the eyes, ears, skin, etc. Among them are the eye strains, the ear strains, the neck strains, all conditioned by some defect in the symmetrical balancing of other muscles of the body.

These pains are usually in the frontal and occipital areas, and are often extremely persistent.

So far as statistics now lead us, the eye headaches are the commonest; they are located either frontally, when the chief irregularities of tension, with, therefore, a compensatory overstrain to correct, are in the eye muscles themselves, emmetropias, asthenopias, astigmatisms, hypermetropias. After years of compensatory overactivity of certain muscles, a real occupation neurosis may set in in the overstrained muscle group, showing itself in pressure over the eyes, frontal headache, and at times various other spasmodic or neuralgic phenomena.

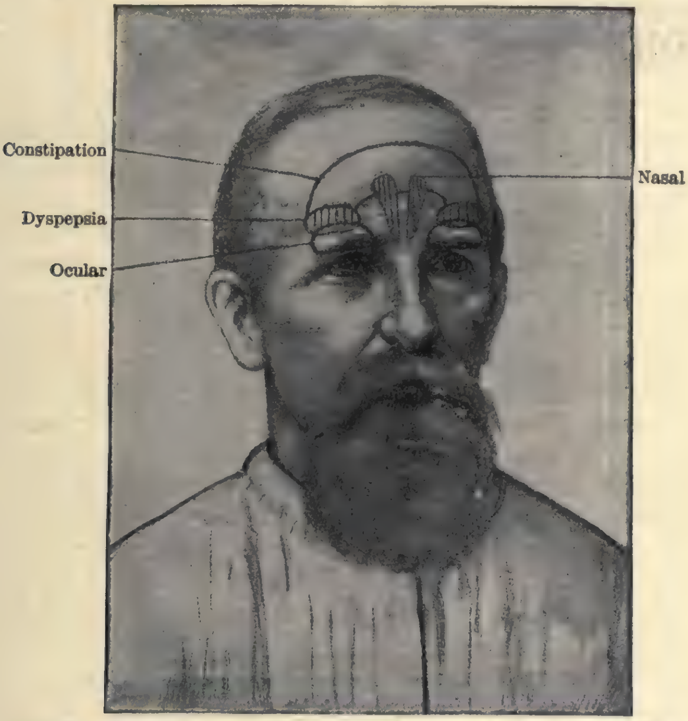

Fig. 2.—The Causes of Localized Headache, According to the Exact Site of the Pain. (Butler.)

Another group concerns the entire position of the head, which not infrequently is held asymmetrically to compensate for unsymmetrical picture formation in the eyes. Here there develops, very frequently, an occupation neurosis in the chief muscles that support the head, with stiffness in the neck, and deep-seated, sometimes parietal, headache.

Desk workers, literary workers particularly, are victims of this type of headache.

Frontal headaches of a related nature are found in many individuals whose corrugators are constantly contracted—photophobics.

Oncoming deafness, asymmetries of hearing, possibly of smell, may account for certain headaches of this type. And, furthermore, it is certain that asymmetries of posture, disproportion in the body equilibrium, can produce similar headaches, chiefly occipital. Certain experiences in department stores, factories, etc., have demonstrated the tendency for certain positions to develop this variety of headache. When the nature of the work was modified, so as to permit greater freedom, and a more evenly balanced activity, right and left, forward and backward, the resultant freedom from headaches has been remarkable. The backaches and headaches of factory hands, seamstresses, shop girls, and others working in positions in which the factor of asymmetrically balanced muscular activity is constant can be in large part explained.

Not all of these workers have such disorders, but they are frequent. Neither is it true that all of the headaches of the nature described are due to these asymmetries of muscular activity which entails constant stress upon one set of muscles. For one to claim them all to be due to eye strain implies a mental squint which, if sufficiently long operative, will bring about an occupation neurosis of the logical faculty—as a mild affection—a faddist of the more intense grades, a crank—in superlative terms, a victim of a delusion. Unfortunately, all these grades exist; the charlatan, a fourth, fattens on the teachings of the other three.

The treatment of these headaches is often very brilliant; by proper glasses, by proper adjustment of gait and position, and by correct teachings, exercises, and placements in the various occupations. Each situation must be met by a complete analysis of the motor habits of the individual and their correction. In reaching a judicial estimation of any cure, careful consideration must be given to the factor of faith, which is a very subtle and important element in all therapeutics.

Acute, subacute, and chronic inflammations of the eye structures occasionally give rise to severe headaches. These may follow conjunctivitis, iritis, keratitis, and particularly glaucoma. The latter is of special importance in its differentiations from neuralgias.

The therapeutics are specific for the disorder in question.

Nasal and Frontal Sinus Headaches (*see Fig. 2, previously referred to*).—The trigeminus is often markedly affected in nasal disorders, either mechanically or reflexly. Swelling of the nasal mucous membrane results in the well known heavy feeling in the head due to a nasal catarrhal inflammation. Acute or chronic inflammation of the frontal and accessory sinuses almost invariably produces headache. In acute frontal sinus disturbance there is a distinct frontal headache, sharply localized between the eyebrows and often very painful upon pressure, sometimes even to touch. Pressure over the malar bones may be painful in catarrhal inflammation of the accessory sinuses. Transillumination reveals the swollen membranes, or the filled sinuses. In purulent cases there is frequently an extension of the pain area, and pulsation is not uncommonly felt.

Polyps in the nose are responsible for some reflex headaches. Obstructions which cause a disturbance of the circulation cause headaches in the frontal areas.

In the nasal sinus cases there are usually accessory signs, which require specialistic examination.

The therapy must vary according to the cause. In the acute catarrhal cases small doses of atropin, combined with aconite, with local astringent and emollient sprays, are often sufficient to bring about a cure. In the influenzal types salicylates can be added to advantage. Surgical measures are necessary when there is pus; polyps or other obstructions are to be removed, care being exercised to avoid ethmoid infections.

Ear Disease Headaches.—These are often combined with earaches or with mastoid pains. The pain is parietal, often widespreading, and is usually increased by jaw movements. Extension to the mastoid, with pain on pressure, is always to be watched with care, since brain abscess often shows itself by such signs, following middle ear or labyrinth disease.

Labyrinthine disease gives rise to headaches of the same nature as those encountered in middle ear disease. In addition one encounters labyrinthine nystagmus—rotatory, horizontal—vertigoes, cerebellar gait, and other symptoms of disturbance of space coördination. An exhaustive application of the Barany tests is needed for the purposes of diagnosis (see Vertigo).

The treatment is usually palliative when the disorder is purely serous or catarrhal; aconite, belladonna, heat, or surgical when there is pus.

Bone and Periosteal Headaches.—These are not frequent. They follow trauma, or are due to caries or gummata. Their localization, tenderness to pressure, and palpation features are usually sufficient to afford a clew to the diagnosis.

Here the treatment is etiological.

Myositis or Indurative Headaches.—This is a very distinct form of headache, not uncommon, frequently mistaken for migraine, often of a subacute or chronic nature, and readily cured by appropriate methods, although at times requiring a comparatively long period. Swedish investigators particularly have studied it, and it has been of late much investigated by Cornelius, Peritz, and Edinger, the former two allying the headache with neurasthenia, for which there is no proof.

I speak of these as myositis headaches. Edinger calls them indurative headache, because careful palpation of the muscles of the head, particularly at the tendinous aponeuroses of origin or insertion, reveals slight thickenings or nodular hardenings, which are often excessively painful to the touch, and in which tenderness is present, not only during the height of an attack of pain, as may be seen in migraine, for instance, but remains permanent.

The localization of the more important of these painful points is seen in the accompanying diagram.

These correspond, in part, to the Valleix points, at one time much discussed, and now frequently forgotten.

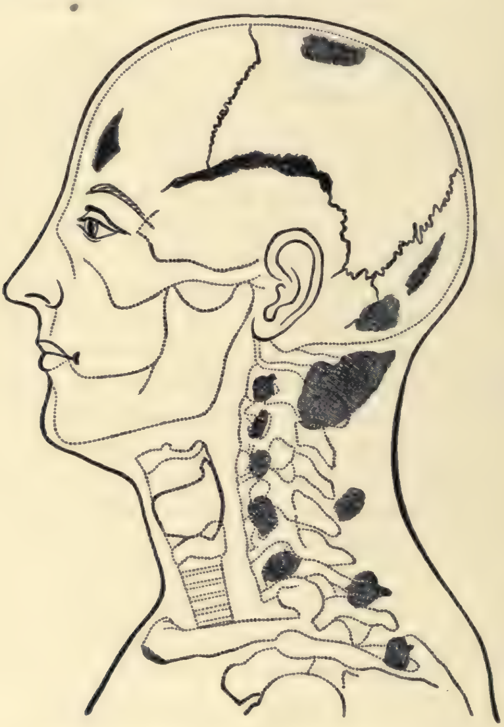

FIG. 3.—LOCATION OF INDURATIVE MUSCLE HEADACHES. (Edinger.)

This form of headache is very frequent in women. At times it is extremely severe, almost resembling an attack of meningitis, with agonizing pains in the occipital region and nape of the neck. At times there is pronounced nausea from the agonizing pain. There is no elevation of temperature. On palpation, the forehead, parietal and temporal regions are rarely hyperesthetic, as is so frequently the case in true migraine. At the insertions of the muscles at the back of the head there are excessively tender points, and at times one finds nodular swellings in the belly of the muscle, or at its insertion, sometimes multiple, which by deep massage may be in part dissipated.

In the chronic cases the headaches are apt to be persistent, occipital, with periods of remission and acute exacerbation. In this period of increase the attack is often like a migraine, may be one-sided, is more often occipital, but may be frontal and occipital, but there are rarely any vasomotor phenomena, auras, etc. Moreover, the pain frequently radiates into the deltoid, which is not common in true migraine.

One feature of these headaches which should lead one to suspect the diagnosis is that other muscle groups are often also involved with pains, stiffness, and diminished activity; similar painful nodules and points are found in them as well.

Exposure to cold seems to bear some etiological relationship to the affection; hence, it is frequently spoken of as rheumatic, which is naturally to be interpreted in the lay sense. Some have expressed the idea of swollen sympathetics, neuralgias, and others speak of uric acid deposits, results of autointoxication. Quack masseurs often speak of these as chalky deposits, which they can rub away, but the exact pathology is uncertain; but deep massage is certainly curative.

The treatment, then, is primarily by massage, which should be begun shortly after the acute period has passed.

The patient should have a laxative, 15 gr. of aspirin, or other salicylate preparation, hot cataplasma, hot bath, and then the sore muscles should be massaged toward the body, usually with the thumb, and at first gently, but later more and more firmly. The nerve trunks are at first gently massaged, but after the second or third treatment deep, strong pressure may be applied with a vibratory motion. A mechanical vibrator may be used later to advantage.

A séance should begin with five minutes, later extending it to 15, and it usually requires biweekly treatments for from two to six weeks.

General measures—tonics, etc.—may be required in some patients who have become below par. Warm climates, free from sudden changes, are helpful, but the best treatment is by means of the massage mentioned.

The osteopaths have helped many of these patients, because the nature of the malady has been forgotten by most doctors of the scientific era. Valleix and his contemporaries described the headaches many years ago. Their modern rerecognition has come chiefly through the Swedish sources.

Headaches Due to Intracranial and Extracerebral Causes

Three large groups may be distinguished:

(1) Those due to meningeal disorder or irritation.

(2) Those due to new growths, causing pressure within and without; and

(3) Those due to disturbances of circulation, or disposition of the blood, or of the cerebrospinal fluid.

Meningeal Headaches.—The headaches of acute meningeal disorder need not be considered in extenso in this place. In cerebrospinal meningitis, tuberculous meningitis, acute meningitis, from extension of pyogenic foci—traumata, middle ear, or sinus disease—the headache is very profound, and occurs early in the disease, before other symptoms indicate the real underlying factors. Certain patients with poliomyelitis complain first of a headache.

These headaches are usually both frontal and occipital. In tuberculous meningitis they are in the neck and the occiput. Other symptoms, however, fever, stupor, stiffness of the neck, convulsions, paresis, etc., soon remove these headaches from the category of those here to be more specifically dealt with.

It is to those headaches due to a subacute or chronic disorder involving the meninges, and which appear prominently and usually alone in the symptom picture, before development of other symptoms, that particular attention will be directed at this time. These are the headaches of pachymeningitis hæmorrhagica interna, pachymeningitis cervicales hypertrophica, serous meningitis, syphilitic meningitis.

In hemorrhagic pachymeningitis the only symptoms may be a localized, diffuse, or persistent headache, with possibly some eye changes, swollen discs, or choked disc. The diagnosis may be impossible without skull puncture, and is only suggestive if the common exciting causes may have preceded, alcoholism, trauma, general paresis, or senile dementia (Blackburn, 1).

Lumbar puncture has been of value in some traumatic internal hematomata.

Serous meningitis, which frequently is secondary to a purulent meningitis, frontal or accessory sinus disease, labyrinthitis, give rise to severe headaches, which resemble those of a brain tumor, brain abscess (q. v.). Choked disc is an early sign. The headache frequently shows improvement on operation on the ear, mastoid, nasal sinuses, and is often relieved by lumbar puncture.

Syphilitic meningitis of the convexity or of the base occasions severe persistent headache, which may come on early or late after infection. In the former case the pain is usually persistent and violent, often definitely localizable on cranial percussion, with attendant skin tenderness. As a rule, general pressure symptoms—choked disc, nausea, vomiting—are absent from the tendency of the disorder to spread superficially. As a rule, cerebral signs appear later, irritative epileptic phenomena, increased tendon reflexes, sensory signs, tingling numbness, aphasias, etc., occasionally one or more appear early. A widespreading, superficial involvement is very significant of syphilitic meningitis, especially when combined with some apathy, or occasional confusion. Pupillary anomalies are not infrequent—slight irregularities, stiffness to light.

The blood Wassermann is present, the cerebrospinal Wassermann negative, and Phase I Nonne not yet definitely known; cell count variable, usually some lymphocytosis.

In syphilitic meningitis of the base the headache is practically always present. It is very violent, occipital, and not infrequently felt deep behind the eyes. The tenderness to percussion is not infrequently above the eyes. In contrast to meningitis of the convexity optic nerve changes are more frequent. Psychotic outbreaks are not infrequent with a paranoid coloring—at times manic, again depressed. Fever is only very occasionally present; subnormal temperature is not unusual. Polyuria and polydipsia are frequent, and the cranial nerves are frequently involved very irregularly; the olfactory among the others, indicating frontal lobe localization.

The serological changes are similar to those just noted. The treatment is by salvarsan, or by inunctions of mercury.

Tuberculous Meningitis.—Here the headache is an early sign. The disorder occurs particularly in young, poorly nourished children or in adults with other tuberculous lesions. In children there is usually a history of an antecedent restlessness; the children are out of sorts; they eat badly, are peevish and irritable, cross and surly, and their sleep is broken. The headache is usually intermittent at first, fugacious, but later becomes persistent, and is marked by ups and downs in its severity. There frequently are also reflex pains in the abdomen and in the chest. Emaciation is apt to be progressive. This headache may persist for months, with slight afternoon and evening rise in temperature, before the more pronounced symptoms of meningeal irritation occur. In others the development of the more sinister symptoms is much more rapid, within a week or month. These are severe headache, stupor, delirium or coma, and convulsions. These children lie in bed in a semidoze or stupor, with frequent crying and grimacing, marked restlessness, throwing themselves about. Older patients show a dreamy delirium, with constant headache. Other signs of tuberculous meningitis are to be found in the appropriate chapter. After the prodromal headache stage is passed, the interests of this chapter cease.

There is no treatment for the headache *per se*. The diagnosis is to be made and the treatment of the tuberculosis is to be begun as early as possible, but the results are not encouraging.

Some rare forms of chronic meningitis, non-tuberculous, non-syphilitic, and of obscure etiology, give rise to severe persistent headaches often associated with choked disc, vomiting, and indications of a hydrocephalus. They are extremely rare. Oppenheim has discussed them in a special section on Chronic Meningitis.

INTRACEREBRAL HEADACHES

Here may be grouped the great number of headaches due to chronic encephalitis of pyogenic or toxic origin, to brain abscess, brain tumor, or arteriosclerosis.

Here the pain may be due to meningeal irritation, as in the meningoencephalitis of syphilis, of paresis, of multiple sclerosis, of alcoholism, lead, etc., or the pain is purely a pressure phenomenon due to a new growth, tumor, abscess, etc. The pains of arteriosclerosis, softening, etc., are certainly not explained on the hypothesis that the meninges alone are capable of receiving painful impressions. Since central sensory pains are known for the extremities in thalamus lesions, it is probable that there are central sensory pains for the fifth and other sensory cranial nerves giving rise to deep or superficial headaches. Their central representation has not as yet been satisfactorily cleared up (Müller, 2).

Brain Tumor.—No attempt is here made to distinguish the forms. Glioma, endothelioma, tuberculosis, gummata, abscess, echinococcus cysts, teratoma, angioma, etc., may all give rise to headache, after they have reached a size sufficiently large to exert pressure. Headache is almost universal in brain tumors, yet it should not be forgotten that it may lag behind other symptoms which definitely point to tumor, or may be absent almost throughout the entire course of the growth. Hard compact tumors are apt to induce headache earlier, and it is usually more persistent, whereas softer tumors, such as infiltrating gliomata, cysts, myxomata, chordomata, by reason of their softer consistency, permit of much molding or adjusting, and pressure symptoms are usually delayed.

The headache of brain tumors is usually very severe and is persistent. It rarely intermits, save, perhaps, in the earlier stages, and even in the free intervals a certain heaviness is usually left. It usually continues during sleep, and is rendered more acute by jars and by sudden movements. Any act that tends to increase the cerebral tension augments the headache: defecation, coughing, sneezing, taking of alcohol, smoking, etc.

The localization varies considerably; usually more or less general or universal, it may be (though rarely) sharply restricted to the general site of the new growth, frontal, parietal, occipital. It may also be found that widespread, heavy headache may be combined with a local, sharply defined one. To trust to the site of the pain as a certain localizing sign is precarious. Many pontine, and especially cerebellar, tumors give marked frontal headaches. Again, other cerebellar growths show exquisite occipital pains and painful percussion points.

Percussion of the head should never be neglected in studying brain tumors with the view to their localization. It is of general rather than special localizing value. - Abscess and cysts frequently show very sharp local percussion tenderness.

From headache or from local tenderness alone a diagnosis of new growth of the brain should not be made. To it the symptoms of choked disc or optic nerve changes should be added. Here again both pain and nerve changes may be absent, and still there may be a tumor—this is not infrequently seen in infiltrating gliomata, and occasionally in frontal lobe tumors. Temporosphenoidal tumors run their course without much headache at times.

The general signs of brain tumor, nausea, vomiting, motor pareses, sensory anomalies, psychical anomalies, these render the diagnosis certain, and often permit of accurate localization.

In cerebral abscess, in addition to the pain, which is often very intense, the antecedent history is all-important. Given a trauma, a suppurative middle ear affection, a suppurative frontal sinus disease, when one finds a persistent, intense pain developing, with temperature elevation, a brain abscess is probably commencing. This may or may not be accurately localized by percussion.

The therapy is exclusively surgical, save in the case of a gumma, where an antisyphilitic treatment is indicated in the presence of a positive Wassermann of the blood, a positive Phase I Nonne, and possibly a leukocytosis in the cerebrospinal fluid, which latter will be negative to Wassermann.

Headache and choked disc, it should be borne in mind, may be present in multiple neuritis, in lead encephalopathies, in nephritis, in chlorosis, and these should be ruled out in the diagnosis of a tumor headache.

Hydrocephalus.—An increase of cerebrospinal fluid may result from a variety of causes, from inflammation of the ependyma, to blocking of the aqueduct, or to pressure on the veins of Galen. Such an increase of fluid within the ventricles, independent of the numerous causes, will cause intense headaches. With congenital hydrocephalus we have nothing to do.

The headaches of acquired hydrocephalus are usually very intense, but are subject to great fluctuation in intensity. The irregularity of remission is often strikingly characteristic. With the headache are found the general symptoms of intracerebral pressure, choked discs, nausea and vomiting, hebetude, comas, diminished attention, and, finally, various paralyses. Swelling of the head, exophthalmos, may be present. The percussion note is at times modified.

TREATMENT.—As far as possible, the original cause should be ascertained. Surgical removal of a tumor or a cyst, antisyphilitic treatment of a syphilitic ependymitis, tapping the ventricles, lumbar puncture are all to be tried, in addition to hydrotherapy, hot pack to the head, and other agents calculated to lessen serous exudation.

The lumbar puncture may be repeated several times, or the trocar may be allowed to remain, permitting a few c. c. to escape every minute or so.

HEADACHE SYMPTOMS OF TOXEMIAS OR GENERAL DISEASE

Toxemias.—Chronic lead poisoning, alcoholic poisoning, nicotin, arsenic, iodin, iodoform, copper, opium, carbon disulphid, and several other toxic substances cause acute or chronic headaches. In the lead encephalopathies, often complicated by severe nephritis, the headache is usually diffuse; in the milder cases described as a pressure or heavy feeling, resembling the neurasthenic types of headache; in the more severe forms the headache is extreme, and is associated with signs of mental hebetude, at times convulsive movements. The gum line, albumin, changes in blood vessels, the colic, the basophile granulations in the blood, neuritis signs, etc., all help in the diagnosis.

Here the therapy is directed toward prevention for lead workers. Greater cleanliness is the first requisite. In lead mines special masks must be worn. In those in which the lead gains access through the stomach the use of very dilute sulphuric acid—which should also be free from lead—is recommended. I have had no personal experience with this remedy. Hydrotherapy is essential to aid elimination, and a fat, protein, and iron-rich diet is advisable for the reconstruction of the blood cells, the nutrition of the altered nerve cells.

In chronic nicotin poisoning, particularly in excessive cigarette smoking, occipital headaches are frequent. These headaches are frequently associated with pressure signs, and like other toxic headaches result from neural and blood pressure changes.

Acute alcoholic indulgence, acute morphinism, are associated with severe frontal headaches. In the former an intense hyperesthesia of the scalp is very characteristic. In the latter a basal occipital headache is not infrequent, associated with much itching of the skin of the body.

The therapeutic relief is quite obvious, if the toxic materials are still operative; prompt emesis and catharsis should be carried out. Washing the stomach is very grateful. Coffee or caffein is very beneficial in combination with antipyrin or phenacetin and elixir of sodium bromid.

Nephritic Headaches.—These are conditioned in part by toxic factors, in part by circulatory alterations within the brain. They are particularly frequent in chronic nephritis with contracted kidneys.

The pains are usually heavy, rather than sharp; pressure or heaviness is complained of; more rarely acute pain in the forehead.

The presence of albumin in the urine, of diminished urea secretion, of high blood pressure, with other signs of uremic poisoning, of retinitis, point to the diagnosis.

The treatment is that for this form of nephritis, which is discussed elsewhere.

Diabetic Headache.—These usually show as diffuse pressures, with heaviness, with not infrequently irregular neuralgias, or neuritides. The trigeminal is often involved; often there is a neuritis of the arms. Preceding a diabetic coma there is usually an increasingly severe headache.

Sugar in the urine, high specific gravity, thirst, itching skin, and other symptoms establish the diagnosis.

Leukemia.—Heavy headaches are present in this blood disorder. It is frequently associated with vertigo, fainting, and other signs of anemia. Arsenic therapy should be tried.

Anemia and Chlorosis.—These give rise often to intense headaches, especially in adolescent girls. They are frequently associated with trigeminal neuralgias. They are seen in the poorly nourished, overworked factory hands, and all who have the habit of taking the various headache remedies, especially those containing acetanilid, which of itself, in doses over 10-15 grains, has a disastrous action on the iron-oxygen interactions in the red blood cells, thus introducing diminished functional capacity to the already reduced iron content of the cell.

The cause for anemic headaches is not as yet clear, although various hypotheses are advanced, chief of which is the positing of an unknown toxic action.

The headaches are often intensely severe, are continuous, and involve the entire head.

The diagnosis is established chiefly by the color of the patients, their modified condition, and is corroborated by the blood findings.

The therapy is for the underlying condition, which is discussed elsewhere.

Gastrointestinal Headache.—These are frequently of the reflex type already discussed. The headache of an empty and hungry stomach is an example. Various forms of indigestion are similarly accompanied by dull or severe headaches, chiefly frontal.

In hyperchlorhydria severe headache is not infrequent, but more often one finds a sense of malaise and heaviness. It is seen in very typical fashion in the mildly seasick, where hyperchlorhydria and heaviness in the head are frequent.

Many migraine attacks seem to have definite gastrointestinal disturbances as forerunners.

The headache of constipation is classic. It is most frequently a sense of pressure, often relieved by a free stool.

Autointoxication is not a satisfactory answer to the question—Why? Neither can one claim them all to be reflex. Possibly pressure anomalies are at work, in which case the filled venous channels of the abdomen are responsible for the disturbed cerebral circulation. The passage of a large stool cannot cause instant relief from any toxic factor, whereas such a passage has an obvious effect upon the circulation and the visceral sympathetic.

The therapy here is obvious, but, as the treatment of constipation is considered in the other portion of this work, those pages should be consulted.

The headaches of hepatitis, cholangitis, gastroduodenitis are due to infection, to toxemia, and to fever.

Post-infectious Headaches.—Headache is often an obstinate after-symptom of many infectious diseases. This is particularly true for influenza. Post-influenzal headaches are often of the greatest intensity, and when combined with overwork the resulting disability may be extreme.

The headache is usually occipital. It is low in grade, rarely advancing to the sharp ache of a neuralgia. It comes on with the slightest effort that the patient makes to do any mental work. Frequently such patients cannot read a line in the newspaper without evoking a headache which completely disables them. There may be no other symptoms, and the patient does not suffer during sleep, or when walking. Such headaches may persist for weeks, even months.

THERAPY.—Massage of the back of the head, hot baths, frequent feeding, and a two or more weeks' Weir Mitchell rest cure are particularly valuable. At times the patient cannot rest in bed, in which case graduated walks, riding, automobiling are useful. Any tense directive effort causes the headache to reappear.

Free catharsis is desirable. Combinations of bromids and analgesics are at times necessary. Opium is to be avoided.

Syphilitic Headache.—Cerebrospinal syphilis is usually associated with headache, especially when it is at all active. In addition to the other signs of this disorder, the advancing neurasthenia, the pupillary, serological, and cytological changes, etc., which may precede for many months, even years, more patent neurological symptoms, one finds headache.

Even in the period of eruption there may be marked headache. These early headaches are usually occipital or hemicranic; they show a similar tendency to those of the later period, in that they vary in intensity, increasing toward night. During the day the pains disappear almost entirely.

In the later phases of syphilis, particularly in the cerebrospinal types, the headache is more variable. The nightly exacerbations are present, but are not so conspicuous. Syphilitic basilar meningitis has already been noted.

In general paresis the headache is very variable. Many patients complain of a disagreeable pressure in the head, others of pain—but this is usually only in the beginning of the disorder. In the later phases paretics rarely complain of headaches, save at times following a convulsive seizure.

The treatment is to be directed against the treponema.

PSYCHOGENIC AND PSYCHOTIC HEADACHES

Under this head will be classed a large group of headaches. There are those headaches which certain patients develop, either as an habitual selfish reaction, to avoid exertion, or in response to interference with an individual's plans. Such headaches, frequently indulged in, have a tendency to recur at intervals not appropriate to the individual's plans, and contribute not only to enhance personal selfishness, but lack of resistance to more fundamental causes for real headache.

Another large group of headaches is due to the contraction of drug or liquor habits. The patient develops a headache which demands its relief by means of morphin, alcohol, or other stimulant. There is a great deal of this type of headache among the well-to-do classes, as well as in the world of the demimondaine. "Really, my dear, I must lie down, I have such a headache," is the usual formula which precedes dishabille, a dose of drug or whiskey, an erotic novel, and a lazy self-indulgent hour or so.

The treatment of this type of headache as a headache is only attempted by the medical sycophant.

With the almost universal "headache excuse" habit—the usual plaint of all autobiographies, the theme of conversation in the cars, the theater or shopping counter—we shall not deal. Few people go through life without some headache, probably no one has as much as he thinks he has. The headache excuse habit is pernicious; as a symptom of general dishonesty it is revealing; as an efficient excuse it is worn out.

Many so-called neurasthenic headaches are nothing but habit excuse headaches. We slop in our energy, our work, or our efficiency, and we rig up a headache to explain it. Mankind is continually excusing itself for its deficiencies, and the headache is the easiest way out of it, and, as everybody uses the same artifice, it is useless to protest.

Neurasthenic Headache.—Neurasthenic headaches *per se* do exist, but they are rarely found without other concomitant signs. Just as headache with stiff pupils, positive Wassermann, positive globulin and cell count in the cerebrospinal fluid means cerebrospinal syphilis, a headache in order to be neurasthenic must show definite fatigue factors: characteristic ergograph tracings in the muscular sphere, defects in attention, loss of power in addition experiments, and a whole series of psychological reactions, which the work of laboratory workers has established. A diagnosis of neurasthenia should be founded on these alone, and every organic cause should be rigidly excluded. Simon-pure neurasthenic headaches are comparatively rare.

When found, it can be learned by a proper mental analysis that emotional factors play a larger part in the neurasthenic reaction than does so-called overwork. Worry over financial matters, the conduct of children, love affairs, unalterable and grinding bitternesses, economic sordidness, these are a few of the innumerable emotional factors that bulk large in the production of a neurasthenic headache.

The chief feature in true neurasthenic headaches, but which is not by any means sufficiently definite to permit a diagnosis, is the sense of weight in the head. The pain is rarely acute, the head feels heavy; the pressure is usually occipital, but may be frontal or anywhere in the head. It may change from place to place. There are a number of descriptive phrases—iron bands about the head, the weight of a helmet, etc.—but such characterizations have been seen in patients with lead poisoning, nephritis, occupation neuroses, brain tumor, etc. An uncomfortable sense of dizziness or giddiness often seems to pervade the head; the patient says he cannot think—everything is empty. This headache is usually persistent. It is often worse in the morning, clears up in the afternoon or evening, and is made worse more particularly by much talking, writing, or effort requiring much concentration. Emotional excitement may drive it away, but it returns redoubled in intensity and area of extension.

Irritability is a frequent general sign, and the petty pin pricks of life are borne with little equanimity. Slight exhibitions of temper—often generously called temperament—increase the discomfort.

Hypochondriacal depression is frequently present. Such a depression must be sharply differentiated from that of the cyclothymic or mild manic-depressive psychosis, with its frequently appearing headache complaint.

Treatment.—This is found in the chapter on neurasthenia in general. From the present writer's viewpoint, the general principles of Dubois' reëducation method are of the greatest value in the treatment of neurasthenic headaches.

Hysterical Headaches.—Pure hysterical headaches in the sense of head pain conversions in individuals of the hysterical character are here referred to—not the thousand and one pseudohysterical headaches which have already been designated the headache excuse habit.

The true types are not frequent. There is one form that is almost characteristic. This is the hysterical clavus, or boring pain, usually sharply localized, as though a nail were being driven through the skull.

The features of hysterical headaches and their treatment are here discussed in the chapters on hysteria and on psychotherapy.

Cyclothymia.—In the mild attacks of manic-depressive psychosis one finds a characteristic picture that should never be overlooked, since suicide may take place, much to the chagrin of the attending physician.

These patients are mildly depressed; they refuse to permit their mental attitude to appear too frankly, for fear of being considered mentally disturbed, and therefore enhance their physical distresses. They frequently suffer from gastrointestinal disturbances, and often complain continuously of pain in the head.

Careful scrutiny shows that many of these patients are rather slow in their reactions; they talk and move with less freedom than is their usual wont, explaining it by the heavy feelings in their head and their difficulty in thinking. Intelligence tests—Bourdon, addition, etc.—show not the characteristic neurasthenic curves, but those of retardation. Further anamnestic search will probably bring out other "neurasthenic" attacks; perhaps some periods of busy activity and excessive well-being; not infrequently a frank outburst of excitement of varying duration, or a frank depression—"melancholia over a love affair," "financial worry," etc. The family history may show similar periodic disturbances of a mild or severe grade.

These are cyclothymic attacks, and in the depressed stage the treatment calls for careful supervision. Many of these patients commit suicide. The diagnosis of "neurasthenia" has been a fatal blunder.

Dementia Precox.—Hypochondriacal headachy ideas are very frequent in the beginning of many dementia precox attacks. They also masquerade under the term neurasthenia, more frequently under that more modern symbol, psychasthenia. Here one finds the characteristic beginnings of the habit disorganizations, so well emphasized by Meyer, the shut-in personalities described by Hoch, the predementia features of useless day dreaming and half-baked philosophizing written upon by Jelliffe and others.

With the frank outbreak of the psychosis, treatment is possibly ineffectual. It should have begun in the so-called neurasthenic or psychasthenic phases. The thorough-going psychoanalysis of the Freudian school alone offers any hope of getting at such patients.

TREATMENT OF VERTIGO

What is to be understood by the term vertigo? Giddiness, dizziness, and vertigo are used as synonymous terms, and, lacking more precise definition, will remain so in the common speech. What one patient will complain of as giddiness will be described by another as dizziness, and by a third as vertigo.

Dizziness in its early origin refers to dullness, to confusion. Its early Anglo-Saxon form is dysig; dösig in the Danish, tusic in high German; it was used to include a number of conditions of altered consciousness, such as the dull, confused states in toxic deliria, and in other psychoses, but its original etymological significance has been much modified in more recent years. Giddiness is even a better term; the Anglo-Saxon meant by it a singing, with dancing or whirling, and therefore it more nearly represents or expresses the chief features involved in the true vertigoes, which word itself, derived from the Latin—verto, I reel, I turn—is almost an exact description of the phenomenon under consideration.

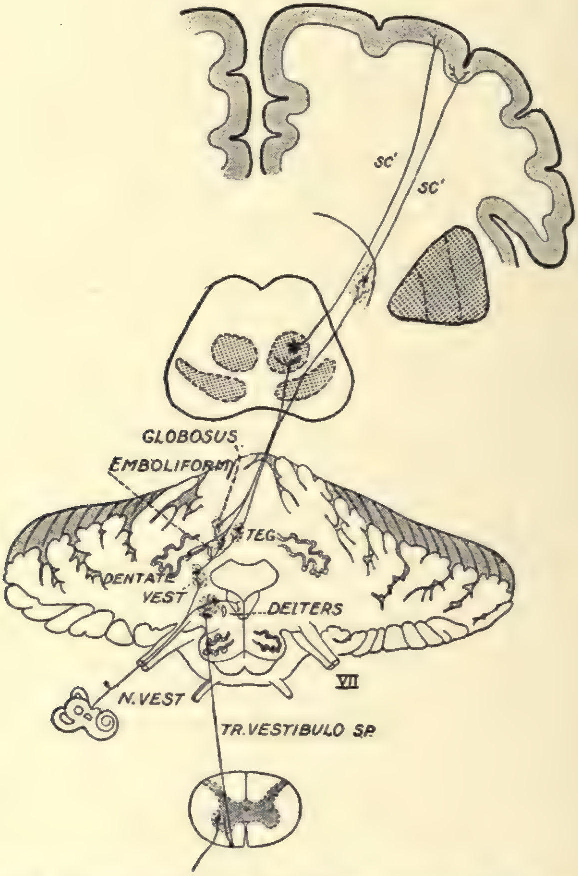

Fig. 4.—Central Paths of the Vestibularis. (Bechterew.)

Vertigo, as here understood, is a clinical syndrome, occasioned by a number of peripheral or central disturbances, each leading, however, to a disturbance in consciousness of the *sense of static orientation in space.*

This loss of static sense orientation may be an isolated phenomenon, in which case one can speak of a pure vertigo; but it is apt to be associated with one or more accessory phenomena, such as nausea, vomiting, nystagmus, pain, deafness, etc., which accompanying phenomena are of considerable importance in localizing the mechanisms involved and in determining the nature of the lesions.

The majority of all vertigoes are labyrinthine in origin, since this organ is the chief ganglion for the perception of spatial stimuli. Probably all true vertigoes are conditioned by some involvement of the paths of the vestibular nerve, whose connections, however, are very intricate and complex. A number of reflex vertigoes are known, but thus far a thoroughly satisfactory anatomical explanation for them, in their relation particularly to the vestibular apparatus, has not passed entirely from the stage of probable inference to that of anatomical proof.

Inasmuch as a rational therapy of the phenomena of the vertigoes must be founded upon the physiological and anatomical considerations—without a knowledge of which no true diagnosis can be made—a brief summary of such facts as are at present available, with some suggestions as to clinical methods for testing disturbed vestibular functions, is desirable.

It has been established without much question that the ear labyrinth is the chief organ of the body connected with the receiving of impressions of its position in space, particularly for the head. The meaning of the physical laws of gravity for the human body is its chief concern. It is adapted to the mechanical stimuli of these laws largely through the otolith organ, which reacts to changes in the incidence and degree of pressure upon its sensory end organs, due to changes in the specific gravity of its surrounding fluids; and also through the semicircular canals which react to changes in position in the three planes of space. The slightest change of the body in space is felt by this apparatus, and in the normally functioning central nervous system any such change is reflexly reacted to by appropriate motor response. This motor response, however, is a complicated mechanism, and all of its elements are not thoroughly analyzed. One of its parts is that of a reflex muscular tonus, by which the ordinary posture of the body is maintained. It is this function that has entitled it to the title of the tonus labyrinth. Just what the complicated interrelations between the proprioceptors of the limbs, muscles, joints, etc., which carry impressions of movements, strains, tensions, etc., and the receptors in the labyrinth may be will not be entered into here. Sherrington (6) has analyzed them sufficiently. The result is the maintenance of the reflex posture of the body, including the compensatory reflexes in the head, and those muscles of the head capable of changing the sense of consciousness of position—the eyeballs in particular.

The labyrinth belongs to a series of organs, then, that work with the physical facts of gravity. It is a part of a great system of connections—which Sherrington has designated a proprioceptive system—which gives the animal, human as well as others, a definite attitude toward the external world, that is, space. It is the most important of these organs. It is connected in a system with other nervous structures performing their part in the same general function, and each segment of the body is caught up in the chain of connections from the lower end of the spinal cord to the frontal area of the cortex.

This whole complicated system of end organs, fiber connections, long and short fiber tracts has its chief center just as every other reflex system has its center. The chief center or head ganglion of this whole proprioceptive system is the cerebellum. The cerebellar connections of the vestibular system, the vestibulospinal, vestibulobulbar, vestibulocerebellar, and, finally, the cerebellorubrocortical components which carry those fibers whose functioning is recognized in the consciousness of space relations are now fairly well known, not in their entirety, but in their main tracts and connections. Hence, disease or disorder which shows any perturbation

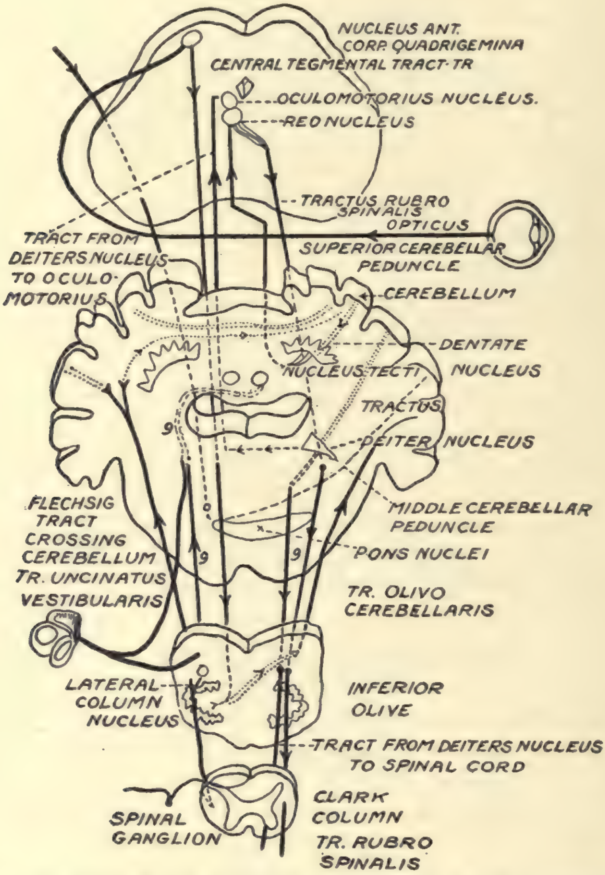

FIG. 5.—SCHEME OF CHIEF PATHS INVOLVED IN RECEIVING SPATIAL IMPRESSIONS AND IN PRODUCING MOTOR ADAPTATIONS TO SPACE LOCALIZATION. (Lewandowsky.)

of the function of orientation in space may be more or less accurately localized along the fiber tracts, carrying the necessary impulses underlying these functions, and an appropriate therapy adopted.

The more precise anatomical description, of which the foregoing is a general résumé, as shown in the work of Obersteiner, Lewandowsky, von Monakow, etc., may be summarized in the following descriptions of the vestibular paths and the accompanying figures of von Bechterew, which show at a glance the chief anatomical features present.

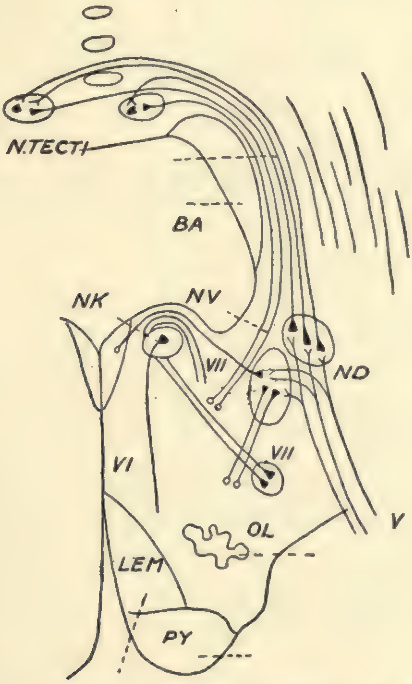

Fig. 6.—Scheme of Central Paths of the Vestibularis. V, Vestibular; NV, Nucleus Vestibularis; ND, Deiter's Nucleus; VC, ascending path from Vestibular to Cerebellum; NT, Tectal Nuclei; OL, Inferior Oliva; PY, Pyramidal Tract; L, Lemniscus; VII, Facial Nucleus; VI, Abducens.

Nervus Vestibularis.—The fibers of the median acoustic root (Lewandowsky—mixed) constitute the central prolongation of the bipolar ganglion cells which make up the vestibular or Scarpa's ganglion. The peripheral prolongations of the cells originate in the walls of the semicircular canals. The thick bundle of the median root pushes its way between the spinal trigeminus root and the corpus restiform (inf. cereb. peduncle) lying at first close to the median edge of the accessory spinal nucleus, and reaches dorsally like the tines of a fork toward the end nuclei. These end nuclei of the vestibular are the triangular and the large cell nucleus.

The Triangular Nucleus.—At the upper exit level of the hypoglossus laterally from the IX-X nucleus there begins a uniform gray area which stretches toward the middle raphe, pressing these nuclei, as the XII nucleus disappears. It has the form of a right-angled triangle whose hypoteneuse is made up of the floor of the 4th ventricle. Cerebrally, it develops more laterally and disappears in the neighborhood of the abducens nucleus (VI). Throughout the entire region there are found disseminated large and small cells developed in a thick network of fibers showing no tendency to grouping, although the cells lie thick at the medial and ventrolateral angles. Further large cells are commoner along the entire ventral border. In Weigert stained specimens the triangularis is dark by reason of the many interlacing fibers. One sees above the median portion the fine fibers of the dorsal longitudinal bundle of Schütz. A fairly circumscribed and constant but very small group of cells lies in the dorsal angle of this nucleus, reaching dorsally and spinally beyond the limits of the triangularis. It is the nucleus-funiculi teretis, and has, so far as present anatomical methods have revealed, only uncertain direct connections with the triangular nucleus.

Large Cell Nucleus.—Under this term is grouped a number of separate nuclei belonging to the end station of the vestibularis. With the beginning of the inferior cerebellar peduncle one finds on the median side a quadrangular area which consists of round crosscut nerve fibers between which nerve cells are distributed (formatio fasciculata). Roller showed that these fibers originate directly from the acousticus, constituting a spinal acoustic root. As one passes cerebrally the area of the gray substance (nucleus descendens) increases, and especially in the upper levels, at which the fibers of the main portion of the median root bend laterally and ventrally, one finds a grouping of especially large cells, which is termed the Deiter nucleus. This lies in the floor of the 4th ventricle in its lateral portions. In the lateral angle of the ventricle, also dorsolateral from these especially large cells, and also reaching into the cerebellum, are small cells which Bechterew has regarded as special endings of the vestibular nerve; the vestibular nucleus, Bechterew's nucleus, nucleus angularis.

Thus the large cell nucleus consists of (1) spinal acoustic; (2) Deiter's, and (3) Bechterew's, nucleus. Cajal describes also a crossed root of the vestibularis whose bundles can be traced along the dorsal border of the spinal trigeminus root through the raphe and can be followed to the other side (Bechterew).

Of the connections of the end nuclei of the vestibularis those to the cerebellum are the plainest. Strong, somewhat swollen bundles of nerve fibers go from the Deiter and Bechterew nuclei dorsally in the cerebellum. Fibers from the nucleus triangularis also join them. The acoustic cerebellar tract lies on the medial side of the inferior cerebellar peduncle, in the medial portion lateral from the sup. cereb. ped. in which a portion also goes.

The majority of the bundles go to the cerebellar worm and end, mostly crossed, in the nuclei of the roof (tectalis), probably also in the nucleus globosus and nucleus emboliformis.

Within the superior cerebellar peduncle portions it may be said that, according to Bechterew and Flechsig, the Bechterew nuclei are connected by means of commissure fibers which pass out with the superior cerebellar peduncle from the cerebellum and bend arcwise in the posterior angle of the crossing of the superior cerebellar peduncles.

Of the further connections of the vestibularis the following may be said:

(1) The Triangular Nucleus.—From the median angle of the triangular nucleus there go numerous, but not arranged in bundles, fibers through the posterior longitudinal bundle through the raphe and tegmental areas. They probably constitute a central connection of these nuclei. From the entire ventral edge of this area there go numerous isolated fibers which go ventrally in fine groups deep into the substantia reticularis in the region of the cells of the nucleus lateralis medius.

(2) Out of the large cell nucleus strong fibers go in a ventromedial direction, partly crossing through the outgoing root of the facialis to the tegmental region, and here bend between the 6th and 7th nerve longitudinally either cerebrally or caudally. These last fibers belong in the ventrolateral portion of the homolateral and partly heterolateral anterior ground bundle as the vestibulospinal tract. None of these fibers come from Bechterew's nucleus.

Other fibers pass medially and as arcuate fibers go to the raphe and the contralateral tegmentum, and hereby probably go to the brain cortex.

The addition which the posterior longitudinal bundle receives from the large-celled nucleus is of importance.

Further, it is easy to find fibers which go from the Deiter's nuclei into (or perhaps only through) the abducens (VI) nucleus, also not a few fibers join the corpus trapezoides which originate from Deiter's nucleus.

Symptoms.—Pure vertigo consists in the loss of the sense of static equilibrium, and shows itself in a variety of ways and in varying degrees, according to the individual and his attitude in space—standing, lying, moving, etc.

In the milder grades one has the sensation of uncertainty, one fears one is about to fall, forward or backward, or sideward. The slight reel of the intoxicated individual, while rarely a vertigo, is conditioned by impairment of sensations of the joints and muscles which is part of the proprioceptive reflex arc already alluded to. Other patients feel as though they were turning about in space, others that space is turning about them; objects are rotating about them and they are still, or they are rotating and the objects are still. The optical illusion, so called, of the sense of individual movement experienced by one who is seated in a non-moving train while a train is moving alongside is an illustration of this type of vertigo, but here only the cortical and optic fibers are involved. In some

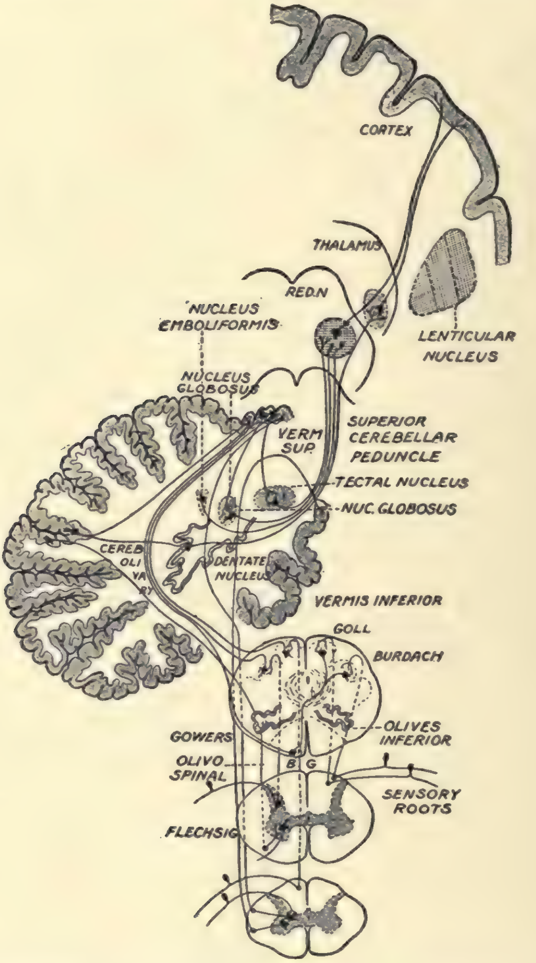

FIG. 7.—SCHEME OF INCOMING SENSORY FIBERS SUBSERVING STATIC EQUILIBRIUM. (Bechterew.)

circular panoramic shows with a rotating canvas one obtains true vertigo sensations. Rapid rotation on a piano stool will induce a rotatory vertigo.

Or the patients may have only a sense of unreality of their position sense; they may be swimming or floating in the air; consciousness is confused and unable to record any focal points.

Various vertigoes show on effort to walk. The patients sway, the reflex attempts at adjustments are underefficient or overefficient, the patient is steered to the left or to the right, or forward or backward, or makes menagerie movements, or irregular zigzags, first in one direction and then in another—all conditioned by the disturbed efficiency of the reflex apparatus whose inadequacy is constantly recorded in consciousness by the sense of falling, and often conscious efforts at repair are the occasion for the overcorrection or the undercorrection of the defect. This is often true in cerebellar cases, which may show the classical "drunken" gait. The vertiginous retro- or propulsions of the paralysis agitans patient are other illustrations of a conscious interference with one of these equilibrium disturbances. This is allied to the forced movements.

Accompanying phenomena are numerous and diagnostically important. Nausea, vomiting, weakness, unconsciousness, pain, roaring and buzzing in the ears, deafness, ataxias, incoördinations, asynergias, adiadokochinesis, disturbed reflexes, tremors, forced movements, nystagmus, convulsions, blindness, mental deterioration, etc.; these, singly or in combination, are among the many accessory phenomena which may be associated with vertigo, and which by their combination determine the diagnosis.

Clinical.—We have limited our conception of vertigo to some involvement of the vestibular portion of the proprioceptive system, either peripherally in the labyrinth or centrally in the extra- or intracerebral or cerebellar paths and connections of the vestibular nerve. Certain vertiginous sensations, however, are met with which are allied but remotely with this conception, and these had better be dealt with before approaching the chief vertigoes.

THE VERTIGOES OF IMPAIRED CEREBRAL CIRCULATION.—Here dizziness, faintness, and partial or complete loss of consciousness are often accompanied by vertiginous sensations. Here there is a loss of consciousness of general space relations which is not related to those of static coordinations, as it should be in true vertigo, but is a part of an impairment of general consciousness.

Grave anemia, cardiac defects, severe pain, these may occasion the vertiginous attacks. They do not properly belong in this subject, and their therapy must be sought under anemia, hyperemia, heart disease, etc.

REFLEX VERTIGOES.—A number have been described, but if a complete examination of the labyrinth be made it has been found that many can be referred here. This is true particularly of the gastric vertigoes so widely described. Certain vertigoes are frequently associated with a chronic sclerotic gastritis in which there is marked constipation. The mechanism of these vertigoes is not understood. They are most frequently termed toxic vertigoes—from unknown and hazy indefinite toxemias. Vomiting, which is often intense, is known to modify the pressure in the labyrinth, which, in turn, may account for the vertigo in those gastric cases not accompanied by any aural complications (Raymond, 5).

Arteriosclerotic Vertigoes.—These are usually associated with hypertension, which hypertension involves the cerebrospinal fluid and the labyrinthine fluids (Lafite Dupont, 3). Many of the so-called arteriosclerotic vertigoes are associated with tinnitus, loss of high-pitched tones, and diminution in bony conduction.

The proper therapy here is directed to a reduction in the arterial tension—at times even requiring lumbar puncture. Iodids and chloral are useful. It is doubtful that a rigid dietetic regimen has much effect. A definite withdrawal of excessive calcium in the diet may possibly retard the calcification, in which case the familiar bread and milk, ideally prescribed by dietetic savants for the aged parent, would be anathema maranatha.

Toxic Vertigoes.—Here one may consider the vertigo due to certain drugs, such as salicyl derivatives (salicin, quinin, etc.), the nicotin series (tobacco), alcohols (veronal, trional, etc.). Both quinin and nicotin have a specific toxic action upon neural elements such as those of the labyrinth and of the retina—tobacco amblyopia and quinin amblyopia are well-known toxic reactions which are paralleled by the vestibular reactions showing themselves in vertigo. The action of the alcohols on all sensory fibers is sufficient to account for alcoholic vertigoes.

Certain rarer toxic vertigoes are met with in pellagra (here probably a sensory neuritis), in Gerlier's disease—probably associated with involvement of the posterior longitudinal bundles (ocular muscle apparatus).

Vertigoes which follow the acute infectious diseases are uniformly due to secondary complications in the labyrinth.

Vertigoes Associated with Epilepsy.—In certain patients petit-mal-like attacks occur with vertigo, pallor, confusion, partial falling, tinnitus, and sometimes nausea. These cases are often differentiated with considerable difficulty. Examinations of the labyrinth should decide and determine the therapy.

Attention should further be directed to another type of epileptiform convulsions associated with vertigo. These are the cerebellar fits of Jackson and point, with other symptoms, to disease of the cerebellum.

Labyrinthine Vertigoes.—These constitute 90 per cent. of all of the clinical varieties. From what has preceded it is seen that the arteriosclerotic, toxic, and reflex vertigoes may be also labyrinthine.

One of the most pronounced types of labyrinthine vertigo is *sea sickness.* Train sickness is another variant of the same disorder. The individual with rapid labyrinthine reactions is apt to suffer from sea sickness which is nothing more than hyperstimulation of the semicircular canals in which particular the superior canal is the most important. Hence the dramatic effects of a rolling sea in contrast with one in which the boat pitches.

Treatment.—For many patients there is none. They must grin and bear it and hope the boat does not roll. Many have made up their minds to be sick, and sick they will be. With the large modern steamships the terrors of sea sickness have almost disappeared.

Sea sickness has nothing to do with the stomach, and the most elaborate antidietary arrangements have little effect. The prospective ocean traveler should pay little or no more attention to his stomach than common sense dictates. Many prospective travelers fail to remember that cold, damp weather is not unusual on the ocean at all times of the year, and fail to be provided with warm garments. The chilliness due to a wabbly vasomotor control—through the labyrinth—and that of the ocean is one of the bugbears of the sea-sick individual. Warm clothing will remove one element of this.

Rooms about the middle of the boat are preferable, but end rooms are rare in the new vessels. Plenty of circulating air in one's cabin aids in cultivating a normal state of mind to the many smells and stuffy sensations in ocean traveling. Pay no attention to drafts; we strain at gnats and swallow camels constantly in our superstitions about drafts.

Sensitive individuals can gradually accustom themselves to the labyrinthine hyperstimulation by reclining whenever the motion is appreciable, and reclining in such a position that the motion is least felt in the superior canals. Thus they should shift their chairs according to the roll or pitch. When the vessel is quiet it is best for such patients to get about as they would under ordinary circumstances.

It is well for the affected individual to recline from the first—and preferably on deck. One would best keep the eyes shut if the horizon is bobbing up and down, as the necessary constant adjustment of the eye muscles aids in causing sickness. On very bright days tinted glasses help to restrain the glare. An interesting series of books, during the reading of which the traveler can close his eyes and ruminate, is advantageous; too continuous reading is not to be advocated. The playing of cards is a useful and advantageous change. It diverts attention.

In lying down one should lie as flat as possible—semireclining does not so place the plane of the superior semicircular canal as to cause the least flow of fluid possible within it. Half sitting up is as bad as standing up. One flat pillow is about all that one should use.

Inasmuch as it is easier to vomit something than nothing sick individuals should eat. If they lose one meal eat another. Nothing is worse than the endless retching of an empty stomach, and especially one made more irritable by alcoholics—including champagne. The effect of "champagne" is one purely of suggestion, and kept constantly alive by the commercial interests of the dealers. Alcoholic beverages are of no particular value unless one takes enough to anesthetize the labyrinth. Those accustomed to them will be harmed less than novitiates.

One should try to eat immediately upon arising or sooner. Here fruit is useful. It matters little what one eats; those foods most liked are best. Any food not relished when well is best avoided. If one detests clam broth, or meat broths, one should not believe they are panaceas for sea sickness. They are not. The oftener one vomits the oftener one should eat or swallow liquid food.

It is very uncomfortable sitting in a stuffy dining room waiting for one's food. Hence go to dinner just as it is about to be served, and begin to eat immediately. After eating it is a good plan to lie down for a short time and not stamp about deck under the delusion that one is aiding digestion.

The unusual changes in habits are apt to bring about constipation, especially when one eats very little; this is best counteracted by eating more fruit and salads, drinking plenty of liquids, or by an occasional pill of aloes, aloes and mastiche, or similar laxative.

The headache of seasickness is best combated by eating, by coffee, and by small doses of bromids and phenacetin. The widely used headache mixtures incorporating caffein and antipyrin in the elixir of sodium bromid are useful. The sodium salt of veronal in doses of from 8-10 grains, given by rectum in suppository, is a very useful remedy in causing sleep and in relieving excessive irritability of the labyrinth.

Vestibular Vertigoes.—At one time loosely grouped together under the term Ménière's disease, the analyses of later years have shown a great variety of these affections depending upon the anatomical site of the lesions. One must distinguish between (1) disease of the peripheral end organ, (a) partial or, (b) complete—these are the vertigoes of partial or complete labyrinth disease.

(2) Disease of the first neuron, (a) paresis, (b) paralysis of the vestibularis.

(3) Disease of the primary end nuclei in medulla and of Dieter's nucleus. The latter gives a special symptomatology termed Bonnier's syndrome.

(4) Disease in the region of the posterior longitudinal bundle—associated with eye movement vertigoes.

(5) Disease of the nuclear region of the eye muscles in the corpora quadrigemina.

(6) Disease of the pontine eye nuclei.

(7) Disease of central eye paths.

(8) Disease of cerebellum.

In disease of all these regions vertigoes are to be expected by implication of the vestibular nerve; the character of the accompanying phenomenon, especially the nystagmus, determines the location.

(1) In partial or circumscribed disturbance of the vestibular end organs in the labyrinth the vertigo is associated with nystagmus movements. The nystagmus is spontaneous, and shows a long slow, due to the vestibular,

and a quick return movement due to the tegmental nuclei, the direction of the quick movement naming the nystagmus. Vestibular nystagmus usually increases when the eyes are directed in the direction of the quick movement, and usually diminishes or ceases on looking in the opposite direction. There is usually always a combination of horizontal and of rotatory nystagmus. Barany states that every other form of spontaneous nystagmus is of intracranial origin. If the nystagmus movement is rotatory and horizontal it must be determined whether it is peripheral or central. A peripheral nystagmus to the right should show on caloric, pressure, rotation tests that the right vestibule is functionally active. Should such tests show an inactive right vestibular then the nystagmus must be of central origin. If the right vestibular is active then continued observation of the nystagmus will alone determine. Should the nystagmus continue uninterruptedly 24 hours or more it is of intracranial origin. If it lasts a shorter interval, is interrupted by quiet intervals, it may be either peripheral or central. When there is nystagmus of the well side as well which lasts about two weeks, gradually decreasing, then a peripheral disturbance seems certain. Intracranial nystagmus is not so apt to diminish.

The Ménière-like attacks are either mild or marked. Buzzing in the ears is rare in the mild attacks. There is no impairment of hearing: In the severer attacks there is little buzzing, but hearing is apt to be impaired. In free intervals the nystagmus diminishes or disappears, the Barany caloric reaction is diminished on the sick side.

Total destruction may be acute or chronic; the latter may show no symptoms. The former sets in with violent vertigo, nausea, vomiting. There is marked horizontal and rotatory nystagmus of the well side. The slightest movement of the head increases the vertigo and nystagmus during the first 48 hours; the latter gradually disappears in three to four weeks. There is marked loss of incoördination, with tendency to fall to one or the other side. After the period of quiescence of the nystagmus caloric and rotation tests show the defective function. The galvanic reaction is not usually affected.

(2) Disease of the vestibular nerve [usually due to tumor of base (acoustic, cerebellopontine angle)] leads to similar reactions. Here, however, there seems to be a difference in that Neumann has found that the galvanic reaction is reduced or lost, according to a partial or complete destruction of the vestibular ganglion. Other cranial nerves are here involved as a rule. The cochlearis is frequently involved. Complete deafness does not result. The trigeminus is also often involved and pain, paresthesiæ, or motor defects appear. Cerebellar symptoms may also complicate the picture. The nystagmus is apt to continue in intensity with tumors, and may be on the sound as well as the affected side.

(3) Involvement of the nuclei (encephalitis, abscess, syphilis, tumor)

brings about similar attacks of nausea, vomiting, vertigo, and nystagmus. The symptoms continue and increase as a rule beyond the three weeks ordinarily seen in the labyrinthine disease.

The method of continuous observation aids in locating the diseased focus.

Bonnier's syndrome—due to implication of Deiter's nucleus and contiguous structures—usually causes a marked attack of nausea, vomiting, vertigo, and nystagmus with buzzing in the ears and deafness (Ménière's syndrome), with irradiations to the ninth and tenth nerves causing anxiety, tachycardia, and hemiplegic weakness. The trigeminus and oculomotor are also apt to be involved. Bonnier has also described peculiar somnolent attacks accompanying his syndrome. Little can be done for these cases unless the focus is of syphilitic origin.

(4, 5, 6, 7) Here vertigo and nystagmus are associated in various ways, but the vertigo disappears on closing the eyes. Here forced movements, conjugate deviations, and various skew deviations afford a clew to diagnosis. Caloric and other tests determine the integrity of the labyrinthine functions.

(8) Cerebellar vertigoes have a number of special features. So far as the vertigo is concerned they may not be separable from the labyrinthine or vestibular vertigoes. Hearing symptoms are usually absent. The nystagmus is less apt to be horizontal and rotatory, but may be up or down or oblique, and is usually directed toward the affected side.

There are usually also symptoms of a tumbling gait toward the site of the lesion; there are asynergia and usually adiadokochinesia. No real distinction as to the side of the lesion affected can be gained from the fact as to the subjective or objective motion of the objects during a vertiginous attack. Closure of eyes has no marked affect upon the vertigo nor upon the gait. Caloric and other tests determine a normal labyrinth.

Treatment.—Here there come into consideration the surgery of the ear and the surgery of the cerebellum and cerebellopontine angle. All of these subjects are considered elsewhere in this volume. The ear specialist should treat the labyrinthine cases, not the neurologist. Rest in bed, quinin, and the usual medical treatment which shuts one's eyes to the danger of a suppurative labyrinthitis, brain abscess, etc., is folly.

In the apoplectic form of the Ménière's syndrome (hemorrhagic labyrinthitis)—often mistaken for a cerebral, or cerebellar hemorrhage—the patient must be kept absolutely quiet, the eyes should be kept closed, the room darkened, and all noises excluded as far as possible—telephone, house bell, etc., shut off. Ice should be applied to the mastoid. Leeches are at times of value. The continuous vomiting may be in part relieved by swallowing cracked ice. Surgical interference may be called for.

In luetic cases mercurial injections, salvarsan, or inunctions are called for. It may be noted that the acute labyrinthine disturbance which has been known to occur after the use of salvarsan is probably due to the syphilis and not to the arsenic (Benario).

Sodium bromid, chloral, antipyrin, morphin offer the best medical aids in giving relief to these patients in the acute stages.

Aural Vertigoes.—These may be referred to briefly, for although the vertigo is due to pressure upon the labyrinthine fluids the main lesion may be in the external or internal ear (extralabyrinthine). Removal of cerum is the first procedure. A more complete therapy of vertigoes due to intratympanic exudates, suppurative, or nonsuppurative middle ear inflammation, amylosis of the ossicles, adhesions to the stapes, polyps, cholesteatomata, etc., belongs within the sphere of the diseases of the ear.

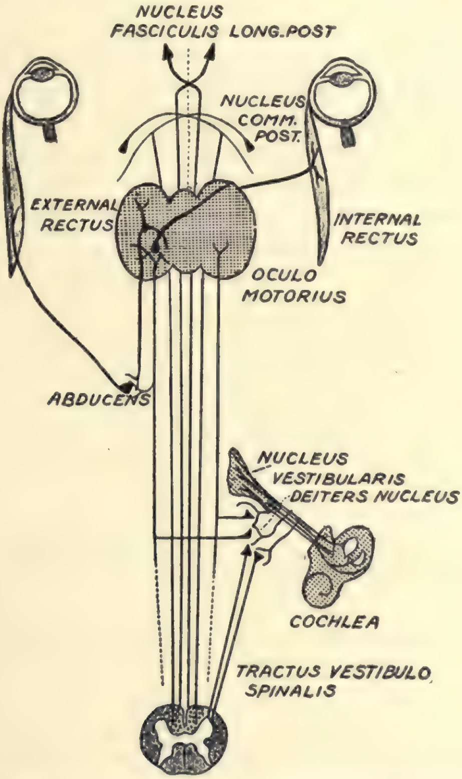

Fig. 8.—Scheme of Ocular and Vestibular Connections.

Ocular Vertigoes.—It has been seen, from the anatomical discussion, that the nervous mechanism of the eyes muscles, by which they are adjusted to binocular vision, and by which the knowledge of the horizontal and the estimation of distance is brought about, is also connected up by collaterals of the posterior longitudinal bundle with collaterals from the vestibular apparatus; as may be seen in the accompanying scheme.

The various illusions, such as moving of trains, etc., may be accompanied by vertigo; car sickness is largely due to the necessity for continuous rapid ocular adjustments, as well as some labyrinthine disturbance. It is best treated by reclining with the eyes closed.

Various errors of refraction, forms of astigmatism, by causing unequal stress of muscular balance, may induce vertigoes. These are relieved by the proper glasses, or operative procedure upon the eye muscles—which latter is rarely called for, save by a few faddists.

Neurasthenic and Hysterical Vertigoes.—The majority of the former are abortive labyrinthine vertigoes, or are due to chronic otitic lesions, which are not infrequent accompaniments in chronic neurasthenic states. The treatment of the ear condition is of advantage both for the neurasthenic fatigue and for the vertigo.

Hysterical vertigoes are rare. Dizziness and mild vertiginous sensations are extremely common, and are reflections of fatigue, mild chronic ear disturbance, excessive use of the eyes with ocular vertigo. That such sensations should be found in hysterical individuals can readily be grasped. Pseudo-Ménière attacks have been described as hysterical in origin. I have previously expressed the opinion that it were better to interpret these vertigoes as due to good and sufficient causes yet unknown than to shut one's eyes to careful methods of examination by calling them hysterical. Vertigo is a not uncommon symptom in the "anxiety neurosis."

TREATMENTS FOR THE EPILEPSIES

Introduction.—Like headache, vertigo, fever, and other general terms, epilepsy, while representing a characteristic and classical phenomenon, is not an entity in the sense that it is always the result of similar causative factors. For this reason science has agreed to speak of *the epilepsies,* recognizing their manifold nature and varying etiological factors.

Such a mode of approach is alone tenable if therapeutic considerations are to be effective. The familiar cry "treat the patient and not the symptom" needs to be reiterated when the subject of epileptiform convulsions is under discussion.

A rigid differential diagnosis, then, is an essential. Such a diagnosis not only should exclude convulsive seizures not due to brain diseases, such as occur in hysterical states, in the compulsion neuroses, etc., but it should also be directed toward a separation of etiological factors within the epilepsy group proper. Thus it hardly needs stating that epileptiform seizures, which not infrequently are the precursors of general paresis, call for an entirely different mode of approach than those due to chronic alcoholism or to multiple sclerosis.

It is here assumed that the great mass of epilepsies are due to definite brain changes. For the most part epileptic convulsions rest upon as solid an organic basis as general paresis or some similar disorder conditioned by brain disease. Yet, it is also certain that transitory changes may take place within the cerebral cortex which may give rise to one or more epileptic seizures and then recede.

The most characteristic of these changes is seen in toxic states, notably in alcohol, where a tissue edema interferes with the normal functioning of the complicated motor mechanism of the brain. Such a related tissue edema is also seen in certain forms of endogenous or autotoxemias—the acid intoxications—such as are seen in defective thymus activities, in defective parathyroid functioning, where it has been inferred that there is an interference with the calcium intake, which in its turn does not combine with the body acids. In experimental thymus animals a well-marked tissue swelling and edema are present in the nervous system. This tissue edema is the cause of the epileptiform convulsions seen in these animals after thymus extirpation.

The question is still further therapeutically complicated when just this general group of cases comes under consideration. The possibility, even the probability, exists that chronic intoxications of the general nature of those just outlined can give rise first to recoverable—then, later, to irrecoverable—tissue changes. A toxic gliosis is set up which ultimately forms a definite pathological alteration, easily demonstrable by histological means, as Alzheimer and others have abundantly proven.

The epilepsies of nephritis, of diabetes, of lead poisoning, of alcoholism need only to be mentioned to be dismissed to their appropriate sections for consideration.

But there are still other conditions which may determine epileptiform seizures of a less sinister aspect, or they may so closely simulate them as to make it extremely difficult to make a separation. In his borderland studies Gowers has laid special emphasis on these phenomena. They include faints and fainting fits, vagal and vasovagal attacks, certain vertigoes—already treated—and certain migraines, likewise referred to in that section.

Certain fainting spells bear a close resemblance to minor epileptiform or petit mal attacks. The more frequent mistake, however, is to regard petit mal attacks as fainting spells. When these are cardiac in origin one rarely finds excitement following the attack, whereas in petit mal such motor manifestations are frequent. Proper cardiac tonics also aid in establishing a normal state and clearing the diagnosis.

Vagal attacks, due to pneumogastric disturbance, often resemble minor epilepsies. There is usually some gastric, respiratory, or cardiac distress, with pain and a sense of suffocation and of impending death. The extremities are usually cold, from vasoconstrictor increase, slight tetanoid spasms occur, with partial clouding, unconsciousness, and a certain mental heaviness. These attacks usually last from ten to fifteen minutes, and may continue for an hour. Moreover the development is gradual. The close relationship of these attacks to the attacks of anxiety neurosis of Freud should not be overlooked.

Put in tabular form, for the purpose of obtaining a quick review, one may divide the epilepsies, according to strict etiological principles, somewhat as follows:

Tabular Scheme of the Various Epileptiform Convulsions

1. *So-called Functional Epilepsies*—not Epilepsies at all:
   - Hysteroepilepsy.
   - Epileptiform Attacks of the Anxiety and Compulsion Neuroses, Dementia Precox.
2. *Epilepsies of Gross Brain Lesion* (Meningeal, Vascular, Parenchyma, or Bony Disease):
   - General Paresis—Acquired and hereditary.
   - Cerebrospinal Syphilis.
   - Dementia Precox.
   - Brain Tumor.
   - Brain Abscess.
   - Cysts, Echinococcus, et al.
   - Pachymeningitis Interna.
   - Syphilitic Meningitis.
   - Tuberculous Meningitis.
   - Serous Meningitis.
   - Bony Tumor Skull.
   - Bony and Meningeal Injury, Traumatisms, Fractures, etc.
   - Multiple Sclerosis.
   - Cerebral Sclerosis.
   - Arteriosclerosis (Senile, Alzheimer's Disease, etc.)
   - Syphilitic Arteriosclerosis.
   - Encephalitides.
3. *Epilepsies of Microscopic Brain Disease,* So-called Idiopathic:
   - (a) Those conditioned by transitory or fleeting, more permanent tissue changes—chiefly acute or subacute edemas—or changes in vascular supply. Acute or Chronic Nephritis (Uremic Diabetes), Metallic Toxemias: notably bromids, lead, arsenic (including 606), Herxheimer Reaction. Other toxemias: Alcohol, CO_2, Blood acting drugs, malaria, parasites—Rabies, etc., Internal Secretions—Thymus, Thyroid, Migraine.

(b) Those due to microscopic alterations following the acute infectious toxemias (Scarlet Fever, Typhoid, Influenza, Measles, Whooping Cough, et al.).

(c) Unresolved factors, possibly toxic: bacterial, chemical, or anatomical.

With such a review in mind the therapeutics become extremely diversified. There is no longer any point to the question, How shall we treat epilepsy? any more than to the question, How much does a house cost in New York? The question must always be answered. Why! is the epilepsy?

Symptom Review.—Having excluded the borderland cases which have nothing to do with epilepsy one can plunge directly into the midst of the epileptic medley. In this group one distinguishes at once at least three series of phenomena which present widely differing aspects, but yet are all constituent parts of the disorder when seen in its fully developed form. These are the convulsive seizures, petit mal attacks, and the psychical equivalents.

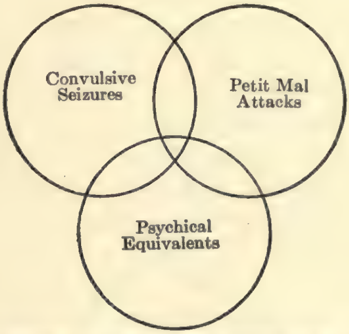

Fig. 9.—General Diagram Showing Mixture of Symptomatic Trends.

The convulsive movements are most stereotyped, and are either general (Hippocratic), or localized (Jacksonian). The petit mal attacks present a number of minor variations to be noted later, whereas the psychical attacks present unusually wide modifications from the slightest increase in irritability to homicidal acts, fugues, and other very diverse symptoms.

As this chapter is not a treatise on the epileptic phenomena only those salient features which are of diagnostic importance will be touched upon.

The Major Epileptic Attack.—The chief features of the attack have been described at length, and with precision since Hippocrates wrote his treatise on "The Sacred Disease."

In the classical major epileptic attack the patient suddenly loses consciousness with or without any preceding warning of sensation of an impending attack (aura). He may cry out, a harsh peculiar cry, then fall, and the muscles of the body stretch out, in irregular progression, in a state of tonic contraction. The fists close, the legs extend, the muscles become tense and rigid, in a sinuous, advancing, and fairly deliberate manner. The face is distorted, and soon becomes livid. Alternations in the tension produce stiff, slow contortions, with oncoming remissions or with progressive shivers. Then a period of convulsive movements follows. Relaxation and contraction take place in rapid alternation, the chest heaves, the body is jerked about, the jaws open and shut. "A clotted mass of motion" is the significant expression of Hughlings Jackson. The lividity increases; urine and feces may be passed, and after a period of a few seconds the patient, still unconscious, ceases to jerk, usually abruptly, and a deep sleep, lasting for a few moments to several hours, terminates a most gruesome performance. On awakening the patient is usually amnesic to all that has occurred, and there are no gross signs of altered motor function, save, perhaps, fatigue phenomena. During the attack the pupils are usually dilated and immobile to light; the patient does not respond to any external stimuli, even the most painful. After the attack there is usually a positive Babinski sign in both lower extremities. The intensity of the amnesia may vary somewhat, as Maeder has shown by psychoanalysis.

This is a very general description of the major epileptic attack. There are numberless variations and modifications in the symptoms when viewed in detail. These can be found in the great monographs of Férê, Voisin, Binswanger, Spratling, Turner, and Gowers, and in the full discussion of the text-book of Oppenheim, Starr, Lewandowsky, and the Osler and Allbutt Systems of Medicine.

MINOR ATTACKS OR PETIT MAL.—These are extremely variable. Many patients will show a preponderance of such attacks—in others they may be rare; in still others only petit mal attacks are known. The proportions are individual and do not allow of detailed statement.

In these slight attacks there is usually only a temporary lapse of consciousness. The patient may halt in his speech a moment, have the sense of being lost, and then go on; they may stagger a moment, and even sit down, or lie down, and then get up. There is usually a premonitory blanching, some aura, or some slight twitching movements of the fingers, or hand, or the muscles of the face. The patient may then be a little dull or sleepy, or may go on with her work or play as if nothing had occurred.

As with the major attacks one finds on reviewing a large number of cases great variation in the minor manifestations, yet the general features are very fixed and significant. The details may be found in the works already referred to.

PSYCHIC EQUIVALENTS.—These are more diverse in their possibilities of variation than the other series of phenomena, and in certain of their forms more constant. Occasionally one finds an individual in whom the psychical equivalents are not known to be accompanied by any of the convulsive or petit mal features. This is rare, however, and can be claimed only after very searching analysis of the patient's life history. Many patients with psychical equivalents will be found to have had petit mal attacks, and the majority have had one or more major convulsive seizures —either nocturnal or diurnal.

The psychical equivalents vary from transitory states of restlessness and irritability to prolonged periods of violent mental excitement. In some a dipsomaniacal period is a true psychical epileptic equivalent. Sudden fugues occur in others, and the patients come to their usual mental state perhaps miles away from home. Some have even crossed the ocean and found themselves in foreign countries among strangers and those speaking an unknown tongue. Homicidal acts may be performed during such an attack. In the "Mad Hercules" of the early Greek poet Euripides such a homicidal act is as strikingly portrayed as in a much more modern text-book of nervous diseases. Epileptic dream states are frequently mistaken for drunkenness.

It would lead too far afield to attempt to outline the numberless variants of the psychical equivalents, many of which are still unrecognized, even by physicians, but two features should be accentuated—these concern the so-called epileptic character anomalies and emotional storms on the one hand, and the more striking epileptic dream states on the other.

So far as the mental features of the epileptic are concerned much might be said. Much more stress should be laid upon these than has been usual. The epileptic character is a fairly stabile thing, the attacks are episodic. Hence, in diagnosis, particular stress should be laid upon the mental characteristics of the individual sufferer from frequent epileptic attacks. In some such character anomalies develop more rapidly than in others, and most recent studies seem to point to close correlations between the character anomalies and the localization of the nervous tissue changes.

While a few epileptic patients show little modification of intellectual capacity the greater number concerned show slowness and difficulty with their mental processes. They grasp questions slowly, and do complicated mental computations with difficulty. Striking results may be obtained by proper use of intelligence tests, as devised by Ziehen and others, from the use of association tests (2), and especially the reckoning tests of Kraepelin.

In some there is a distinct deteriorating process leading to almost complete dementia. The idiotic, imbecile, and debile stages in juvenile epileptics need mention only.

There is often a striking poverty of ideas, which shows up with association tests. This usually leads to an enhancement of the ego of the individual and an increase in the emotional irritability of the patient.

This emotional change is often very difficult to cope with therapeutically. The moody, selfish, contentious, negativistic epileptic is an exceedingly trying problem to manage.

Careful analysis shows that these patients have emotional attacks independent of the more classical symptoms of epilepsy. They have been studied particularly by Aschaffenburg. These patients wake up depressed. They may have had distressing dreams, often sensual in character, accompanied by ejaculations. They become sullen and resistant, look upon the dark side of everything, are reluctant to go to work, or busy themselves about the house. They may begin to tease or to find fault, to grumble and growl, at times are profane. In asylums or sanitaria they want to be let out, and are apt to be violent if opposed or argued with, often attacking with great brutality those who irritate them. Even homicide may occur as a result of such moods, and yet the patient knows what he is doing, and still is irresponsible. Here the legal test of irresponsibility, i. e., of "knowledge of the act and whether it was wrong," is a farce. These patients are incapable of control by reason of a brain disorder: their knowledge of the act is only theoretically impaired.

These periods often alternate with fits of depression and anxiety. The patients are hopeless, never expect to get well, they are tired of life and want to find some relief from their discomfort and sense of incurable illness. Occasionally these patients commit suicide while in these depressed periods.

Attacks of excitement with euphoria and motor unrest are also met with, although this is a much less frequent mood than that of depression. The patient is then expansive, happy, even gay, speaks rapidly, may busy himself much with business things, or about the house, may show flight of ideas. They are often extremely irritable, commit violent acts, swear, or talk aloud for considerable periods.

Some patients develop very typical paranoid ideas, either following a convulsive attack or absolutely apart from the same. They become suspicious, have ideas of reference, of being followed. They refer their inability to hold a position to their being hounded or persecuted, and then they are apt to develop hallucinations of hearing. They often hear their names called—they are threatened. This irritates them greatly, and again one finds another opportunity for apparently motiveless violent acts. These acute, or subacute, mental states usually last only a day or two. They disappear as rapidly as they came. The patients laugh at the idea of their being followed, of having heard threatening voices, and cannot imagine why people should not understand them better, or even assume others to be crazy.

These types of attacks occur in from seventy to ninety per cent. of all epileptics. From the therapeutic point of view it becomes highly important to recognize them in order to protect the patient from his own rash deeds. Often it is necessary to restrain his liberty for a time in order to protect others.

Another series of phenomena has been referred to already. These are the peculiar, and often startlingly weird, epileptic dream states. They are more frequently seen following an epileptic attack; occasionally they precede the attack—as in the case of Hercules, previously referred to—but they may also occur apart from and apparently unrelated to the convulsive seizures.

The simplest form observed is that of dreamlike confusion, which is often accompanied by hallucinations. The patients are able to walk, but they go about in a mild semi-stupor, as though half intoxicated. They see faces, hear voices, smell smoke, or hear bells, and talk about their surroundings as if they were in a daze. They frequently leave their work and commence to drink, or they start a fire somewhere—especially the young patients—or they go into a store and help themselves to anything that pleases them. Kraepelin tells of a patient who had set fire to his bed in order to boil some coffee; others have committed manslaughter in such a mild dream state. Others again are happy and go about in a merry jovial state. Such not infrequently urinate on the public highway, or show their genitals in public, openly masturbate, or may make definite sexual approaches.

After the period of dreamy confusion has passed there is a comparatively absolute amnesia for all that has occurred. Careful research by proper psychanalytic methods may show occasional memory islands, but such are disconnected.

Other dream states show themselves in night walking. Such is to be distinguished from the frequent turning, getting up out of bed, and loud talking of many nervous children. These epileptic patients often perform complicated acts. They get up out of bed, open and close the door, descend the stairs, light the gas, or a fire, rummage about in a closet, and then return and go back to bed after a few minutes or an hour or so. Their movements are highly automatic, they avoid obstacles, although occasionally they walk out of a window and are injured or killed. With certain few individuals comparatively simple conversation can be carried on, poems recited, examples answered, etc. The unobservant lay person may not notice they are practically asleep (La Somnambula).

The severest grades are spoken of as epileptic stupor. The patients can eat and work, but do so as though in a deep drunken stupor—they are without any real knowledge of what is really going on; the amnesia is practically absolute. Their speech consists of a few broken words, or interjections, although occasionally they show continuous rhyming—echolalia or other types of automatic speech. Kataleptic phenomena are occasionally observed.

These dream states vary in duration from a few hours to two weeks—

in rare instances longer—and the course is often characterized by variation in the intensity of the dream state.

One word should be said about the anxious delirium states observed in epileptics. This anxious delirium is one of the most practically important of the epileptic psychoses, as it occurs with comparative frequency in chronic epileptics, and is not associated with convulsive manifestations. It develops within a few minutes, or after a few hours. There is a period of anxious depression, of dreams, of peculiar sensations and haziness. Recurring, almost stereotyped (in the various attacks), hallucinations take place. The patient sees a "black man," or "red blood," or "a devil in a red mantle." Complete disorientation takes place, hallucinations increase, and then anxious delusional ideas develop. He is going to be delivered to the devils—thrown in prison—sent to the gallows, etc. "There is a man outside going to shoot him." "He is being poisoned," and a host of similar frightful ideas with visions reproduce Dante's Inferno within him. He falls upon his knees, prays to God, implores those about him to help him, or at times is scornful, and even bitter, in his anxiety. In this latter state such a patient is often extremely dangerous. He may commit the most ghastly crime. He may run amuck with inarticulate cries and bellow like an angry bull.

Such states persist a few hours, or even a few weeks. The anxious attacks are often mistaken for melancholic states.

A number of variants of these "dream states" are recorded in literature. One is reported by Alzheimer as having persisted 18 months. Such cases, however, are extremely rare and need the most extreme critical scrutiny to pass muster as epileptic dream states—rather than forms of hysteria, aggravation, exaggeration, or simulation.

THERAPY

With this symptom review in mind—especially of the mental features—and with the etiological possibilities before one, one is ready to start on the therapeutic problem.

Naturally when one glances at the tabular summary on p. 632 the suggestion arises that the majority of the particular epilepsies there mentioned either are untreatable or that treatment is of little avail. This is far from being the case, however. In certain of the epilepsies enumerated as due to gross organic lesion the indications for definite therapy are very direct.

Thus, the epilepsies of cerebrospinal syphilis, which latter shows itself in the form of flattened pachymeningeal or leptomeningeal gummata, or as a diffuse infiltrating exudate, or as a specific endarteritis of the terminal vessels of the cerebral cortex—these epilepsies demand prompt antisyphilitic treatment—preferably by from two to five injections of salvarsan intravenously after a complete neurological and serobiological status has been taken. Most of these syphilitic epilepsies clear up as if by magic. This is true particularly of those cases of infantile cerebral syphilis of hereditary origin, with epileptiform convulsions, and at times with various grades of feeblemindedness.

While salvarsan is the ideal spirochetal remedy, other antisyphilitic measures may be employed, but they must be vigorous. Salvarsan should be used when it can be shown that spirochetes are present and active. It is less valuable in other stages of the syphilitic exudates; in rare instances in a cerebral gumma which may show the serobiological reactions.

The epilepsies of brain tumor, or serous meningitis, of chronic bony pressure, of depressed fracture, of cystic formation, of pachymeningitis, and other removable disorders require appropriate surgical treatment. Such surgical treatment is not to be recommended, however, in the absence of definite indications. Indiscriminate surgical therapy for epilepsy is criminal. The chief indications that call for surgical interference are the history of a new growth formation, or of that of an accident, or old injury that might leave residual lesions. A careful neurological status, which points with some degree of definiteness to a particular brain area as being involved, is imperative. Here Jacksonian attacks are of extreme importance in determining the advisability as well as the location, also the presence or absence of aphasia, of disturbance of smell—(uncinate fits)—etc., in which connection it should not be overlooked that new growths of the temporosphenoidal lobes very frequently give rise to very characteristic epileptiform seizures accompanied by hallucinations of smell. Finally the absence of the characteristic serobiological reactions of syphilis—the four phase reactions of Nonne, as they may be conveniently grouped.

Occasionally a general paralysis shows itself first—to the untrained observer at least—as an epileptic convulsion. A careful neurological and mental examination, with the positive Wassermann blood and cerebrospinal test, a cell count of the cerebrospinal fluid of over 10 cells to the cu. mm. and a positive globulin finding, these point without fail to paresis. The neurological status may be negative, even though less frequently no mental defect can be made out on the use of the proper "intelligence tests," yet the serobiological tests have been shown to be almost absolutely positive.

At this phase the proper treatment is more legal than medical. While a prompt medical attack should be made upon the disease it is essential that the sick individual should be so guarded as to prevent the dissipation of his property. Although American jurisprudence has not yet advanced to the point that the four-phase reaction will be accepted as proof of the existence of paresis, the family physician should not neglect his duty in making the interested parties aware of what is likely to happen. He should do all in his power to put legal safeguards in operation to prevent loss and destruction, which are bound to come and, usually, all too quickly. This is preventive medicine of the highest order.

The therapeutic art is dumb in the face of the epilepsies of multiple sclerosis, of tuberous sclerosis, of the various scleroses following encephalitis, etc., so frequently found in the epileptic brain. Here the etiological diagnosis cannot be made—save for multiple sclerosis—before autopsy.

In passing over to the group of the so-called idiopathic epilepsies it may again be stated that they are due to definite brain changes, but that these possibly are not always structural changes, or are structural only in the sense of a change in the protoplasmic character of the cells—such as occurs, for instance, in simple toxemic states.

We are not here concerned with the therapy of uremia, nor of diabetes, nor yet of the metallic poisonings, all of which may cause epileptiform convulsions. The diagnosis made, the therapeutic indications are self-evident.

In the group of the epilepsies due to disturbance of the internal secretions science is not yet on firm ground. It may be stated, however, that certain epilepsies exist which are due to defects in metabolism. Munson's (3) able summary shows the present status. Parhon has gathered the evidence from another angle.

It must be confessed that the therapeutic use of thyroid, thymus, ovarian, testicular, and other organic substances has not given many striking results, but this failure may be due to the fact of its indiscriminate use—the failure to select those epilepsies, for treatment by such means, that are due to metabolic defects. Here the obvious difficulty arises: How is one to determine that the epilepsy is due to a loss or surplus activity of one or more internal secretions? It is only by a process of rigid differential diagnosis, a sharp eye open to other concomitant symptoms of disorder of the internal secretion, and their empirical trial.

Thus in the epilepsies observed in castrated women the therapeutic approach is often evident. It is further apparent that there are physiological castrations from ovarian disease in which a like therapeutic attack would be relevant.

The menstrual epilepsies need to be carefully studied along this same line of thought, and the menopause epilepsies as well.

There are frank epilepsies in subacute or partly developed myxedemas. Here proper attention should be given to the thyroid. Thymus disease is responsible for certain epilepsies.

Finally the general problem of the metabolism of calcium should receive attention, especially in its perturbations due to thymus, or parathyroid disease, and also in its relations to the acids of the gastrointestinal tract. Certain epilepsies show disturbances in the usual calcium interchange, and such epilepsies are much benefited by administration of the calcium salts, or a substance rich in such salts.

General Mode of Treatment

After all is said and done we are still in the position that all the groups of cases, apart from the few that respond to special modes of treatment, arsenic, mercury, iodids, secretion therapy, etc., can be much benefited by general treatment along general lines. These general lines are here indicated at the end of this article, rather than in the beginning, because the epileptic has been too much regarded as a general rather than a special problem. He has been "herded with the bunch," so to speak, and individualization has been too often neglected. He is given bromids, laxatives, and general advice, but is neglected as a scientific problem in individual differential diagnosis.

Prophylaxis.—How far can the various epilepsies be avoided? If the etiological factors referred to in this article are of the importance attributed to them it is seen that the problem of the epilepsies and their prevention extends into the broad fields of preventive medicine in general. Every disease in childhood should receive the best possible treatment, and not even the minor ailments should be neglected, especially if its infectious agent is one that has special affinity for the nervous system. This is particularly true for influenza and for whooping cough, also for syphilis and alcohol.

What can one answer in regard to the question of marriage? This is confessedly a difficult factor. In those instances in which the epileptic attacks are the result of purely accidental factors, trauma, severe illness, encephalitis, it is difficult to see wherein there can be any inheritance of an acquired character. But perhaps there may be factors behind in the individual which have determined an epilepsy in those who have accidentally acquired it. In this case the presence of an epilepsy determiner would be a serious thing to hand down.

The question can receive an answer only by an appeal to experience. Such appeals in the usual statistical studies have only just begun to be of value. Davenport and Weekes (1), in their study, have given us the most searching analysis of this question ever attempted. While it is certain that the material utilized in their study constitutes the worst portion of the community, neuropathically speaking, their conclusions should be carefully considered.

They show from their field study work that such a method for the study of epileptic families, combined with the modern biological methods of analysis of hereditary data, constitutes a vastly improved means of enquiry into inheritance of epilepsy. Epilepsy and feeblemindedness show a great similarity in behavior in heredity, supporting the hypothesis that each is due to the absence of a protoplasmic factor that determines complete nervous development. When both parents are either epileptic or feebleminded most of their offspring are so likewise. Other conditions named, migraine, chorea, paralysis, and extreme nervousness, behave as though due to a simplex condition of the protoplasmic factor that conditions complete nervous development, i. e., persons belonging to these classes usually carry some wholly defective germ cells. Such persons may be called "tainted." When such a tainted individual is mated to a defective about one half of the offspring are defective. When a simplex normal is mated to a defective about one half of the offspring are normal; the others defective or neurotic. When both parents are simplex in nervous development, and "tainted," about one quarter (actually 30 per cent.) are defective. The proportion of tainted offspring is not noticeably higher when both parents show the same nervous defect. Normal parents that have epileptic offspring usually show gross nervous defect in their close relatives. While they recognize that "epilepsy" is a complex, yet they conclude that there is a classical type numerically so preponderant that, in the mass, "epilepsy" acts like a unit defect. They state that their data point to a poisoning in slight degree of germ cells by alcohol, but conclude that the evidence is hardly crucial. That there is evidence that in epileptic strains the proportion of epileptic children in the latest complete generation is double that of the preceding; but there is no evidence that in these epileptic strains the average number of children in a fraternity is greater than in the population at large. The most effective mode of preventing the increase of epileptics that society would probably countenance is the segregation of certain groups of epileptics during the reproductive period.

These conclusions are in need of some modifications. Some of them are not true; thus the epileptic parents may have perfectly healthy children and so may feebleminded. They are overstated, but the general trend is worthy of attention. Should an epileptic patient come into the office and ask advice regarding the advisability of marriage, the answer should only be made after a complete study of the family trees of both parties involved, and of the individual etiological factors in the case under review.

There are epilepsies of so purely accidental a nature that transmissibility does not take place when the stock is healthy. But it is not always simple to draw the distinction between such a purely fortuitous epilepsy and one that appears accidental, but which in reality has been determined by specific resistance absence factors, the accidental causative factor having made a latent possibility effective. When the contracting parties each have had neuropathic taints the results for the progeny will be apt to be disastrous.

But may not marriage still be considered if conception be prevented?

This may be the next form of the question if the physician has offered objections to the marriage of the parties under consideration.

Here other features of an entirely different character enter into the problem. The answer is not simple. In the first place it is not possible always to prevent conception. If conception takes place one is face to face with the question of abortion. The out and out eugenist will not balk at the answer. Whether his attitude should prevail may well be made the subject of an extensive discussion, and every physician meeting this problem must consider it on its own ground in the light of all the facts.

Furthermore the method of preventing conception easiest available, i. e., the wearing of a condom, is for many individuals extremely difficult. Here a choice of two evils must be fairly put up to the individual with a strong accent on the fact that relative celibacy does one little harm after all. For either party an unsatisfied sexuality runs the risk of the development of an anxiety neurosis. This, however, is a much more readily handled proposition than that of an epileptic child or children.

An examination of the tables of Davenport and Weekes (1) demonstrates that the brothel is no solution of the difficulty. It, in fact, is an enormous element in creating the conditions not only in the epileptic camp, but in that of the neuroses and psychoses in general. Man's subjection to the domination of nature's primal instinct and its forcible determination to the sexual object have created the brothel, and with it many of the problems of neuropathic heredity. Hence we should not look to the concubine nor to the prostitute for help in this question.

That reduces us to the duty of inculcating the principles of the sublimation of man's libido if we are to permit marriage. Such sublimations come about through the avenues of religion, of art, of philanthropic work, of the constructive—not the obstructive—type. The cultivation of the pleasures of the mind so dear to ancient as well as to modern philosophers is one of the necessary features in such a program of efficient sublimation, and is one upon which too much stress cannot be laid.

Nature study is an ever ready coadjutor in this fight for a balance between the exercise of the sexual instinct and the forces of an eugenic intelligence. It combines the necessary physical outlet so efficiently cultivated by the Greeks in their gymnastic and out-of-doors exercises with the study of those questions of life and heredity which the individual himself is trying to understand and to solve for his own sake and for the good of humanity.

But turning from the problem of averting difficulties to those more imminent, what must be the attitude toward the children of an epileptic father or mother, or of those where epilepsy simply enters as a possible lurking danger? In one sense these children labor under the ban of a possibility, and even a probability, according to the strength of the absence character in the germ plasm of the parents.

For the many in the population of our large cities the problem has no practical solution. One can speak of it, but what can be done? Nothing! Each case, however, may be made the burden of an appeal to one of the philanthropic group in search of his or her own efficient and saving sublimation.

It is perhaps a pretty dream, but one can hope to see a class of philanthropic workers themselves endeavoring to compensate for the families denied them, by reason of the epileptic burden, by occupying themselves with the children of the class just mentioned.

Just what to advise such workers to do has its difficulties. On the one hand, we may stimulate their interest in the epileptic colonies now in operation, or those planned, or those that should be planned. It is not enough to have such colonies—they must be wisely managed. The board of directors, made up, we may in time hope, of individuals keenly alive to the problems, should work with all their energies to increase the equipment, make life a comfort and strive to eliminate the tendency to routinism that settles upon most community endeavors.

From the organization of "Our Lady of Lourdes," in France, one can learn many valuable lessons in organized charity—both what to do and what not to do; for there is an individualization there that is well worth emulating. It would not be impossible to learn the name and obtain the record of every epileptic in a community if there were philanthropic workers who would throw themselves into such a work. Over one hundred years ago all of the mentally disordered were carefully counted and listed in a Bavarian principality of 70,000 individuals, and an attempt made at that time to learn somewhat of the causes of mental disorder. It is not surprising that Bavaria stands as the leader of the work to-day in this same movement.

Such an individualization is the only way to get at a problem of this type, then, after the need is recognized in its details, an efficient utilization of private and state help is possible.

The various State Charities Societies are doing an enormous work of this nature, and they should be encouraged. Personal experience has led to the opinion that, like most other institutions in this country, that of the philanthropic workers as well shows diffuseness of effort and lack of central organization. That which the Roman Catholic church has been attempting for years in its systematic plan of apportionment in its work might well be emulated in every large community. Religious and philanthropic workers do too much overlapping. They should all get together, apportion their territory, and each cultivate their own garden, and look after the poor and needy in a much more systematic manner. The physician of the country can help immensely in the epileptic problem by preaching and organizing such unification of methods of dealing with this philanthropic work.

The treatment of the average epileptic should be in a proper institution, in fact, the ideal treatment cannot be carried out elsewhere. This becomes apparent in view of the mental state that has been pointed out. There are certain individuals, it is true, who are able to get along in the ordinary social milieu, but they are in the minority. In consideration of the enormous extension of this symptom group, however, there are a great many such individuals, and our treatment may be divided into several sections.

Prophylaxis of Attacks.—General prophylaxis from the eugenic side has already been considered. Here only the constitutional, hygienic, dietetic sides will be touched upon.

Here marked individualization is necessary. It is necessary to take into consideration the social and economic situations, the character of the work of the individual, and the demands made upon him. It has been known for centuries that emotional excitement and mental overwork are disastrous features for this class of individuals, and Hippocrates taught, what is true unfortunately for a few patients only, that a very carefully regulated life may alone be sufficient to bring about a cure in certain individuals. Such a careful regulation is particularly important in the early stages of the illness.

While some modification in the mode of bringing up these individuals may be necessary it is not required that they give up all intellectual work, stop school, etc., etc. A certain amount of training is very essential, but if attacks mount up in frequency under a full day's work it should be reduced to four, three, or even one hour. The work of the day should be planned with regular work and regular pauses.

If the attacks come on in later years an entire modification of the life plan may be called for. Farming, gardening, poultry, and out-of-door work is in general the class of work best suited to the majority of these individuals—due attention being paid to the disadvantages of the summer's heat and excessive cold, and to the dangers from contact with certain tools, horses, wagons, etc., in case of attacks. Many of the institutions for caring for epileptics have carried out the possibilities of industrial cures to fruitful ends, and smaller institutions can well model after them. Nothing is worse for the average epileptic than to be sitting round, doing nothing all day. Journeying from place to place, seeing new scenes, etc., is also bad for the average case.

It should not be overlooked that very frequently, in the very beginning of an epilepsy, especially if it sets in stormily with frequent attacks, absolute rest in bed is good treatment.

The dietetic management of the epileptic is an immense subject. Individualization is here again, as elsewhere, the keynote. Bad tongues, much gas, obstinate constipation, visceral pains, headaches, eructations, these are the frequent gastrointestinal conditions often made worse by unwise or excessive medication. A careful metabolism analysis—in the general sense—is desirable—a carefully selected mixed diet should be experimentally elaborated. Unfortunately chemical research is of less value than actual dietetic observations, which should not neglect the routine of the kitchen and the preparation of the food.

It would be impossible to go into all of the details of such a diet in this article, but since Heberden's day it has been known that, in general, epileptics have fewer attacks on a general vegetable diet than when on a meat diet. This does not mean that such patients should not eat meat, nor that any vegetable is good enough, in fact, experimental evidence shows that certain meats, cooked in certain ways, have no ill effect on some patients, and that, for some, vegetables prove disastrous, especially starchy ones. It must again be stated that much of the gastrointestinal disturbance is due to drugs, chiefly salty solutions.

Regularity in eating is a sine qua non; moderation in eating is equally as important, and deliberation during eating is paramount.

A dietary should be evolved which should give the proper nutritive values, the proper pabulum for the motility of the gastrointestinal canal, and which should contain those ingredients which the individual best handles, as determined by repeated experiment. The chemical and microscopical examination of the feces is of great help in determining this latter feature.

The dietary should be mixed. Its cost depends entirely upon the individual. The average epileptic does best on a fair breakfast, a dinner, and a light supper, eaten two or three hours before retiring. Some patients do better on four meals a day, others on two.

A dietary for four meals, which is more available for private work rather than in institutions, will follow the European custom with first breakfast between six and seven o'clock, consisting of weak tea or coffee, toast and eggs; a second breakfast at eleven o'clock with bread, butter, soft cheese (pot cheese, cream cheese), milk and eggs. A dinner at two or three of soup, meat, fish, vegetables, fresh or canned, and a dessert, chiefly fruits, cooked or raw, depending upon the consistency.

Supper at 7 to 8 o'clock. Rice, milk, with cocoa or a fruit juice.

Some authors say that certain fruits are taboo. Among these are fruits with numerous small seeds—blackberries, raspberries, currants, etc.; cake, pastry, alcoholic drinks. Regarding these it may be said that much depends on the patient and the cook. Light cake is better than heavy bread. The small seed fruit restriction does not particularly appeal to the present writer, unless experience demonstrates their dangerous quality.

Physical Therapy.—Hydrotherapy is of great service for many epileptics. It is not curative in any sense, but is one of the general tonic agents that help to raise the level of nervous energy, and permit gradual useful energy expenditure instead of the purposeless fulminating discharge of a convulsion. To hydrotherapy, mechanotherapy procedures may be added with precisely the same object in view.

Systematic manual training is an important physical mode of treatment. The individual capacities and tendencies of the patient should be taken into consideration in adapting means to ends.

The brickmaking, broom making, printing, carpentering, blacksmithing, and similar industrial occupations, as carried out in many institutions, are ideal goals that make the individual useful, train him for efficient activity, and thus must materially aid in therapy. The development of female occupations should not lag behind that of the men, nor should it include too much needlework or machine sewing.

Pharmacotherapy.—We are not considering the patients for whom special measures are applicable, such as mercury or arsenic in the epilepsies of syphilitic origin. The remedies to be here discussed are used largely for the correction of gastrointestinal disturbances, or are directed toward the depression of excessive motor activity, in order to reduce, if possible, the number and severity of the convulsive phenomena.

Some attacks may be aborted by the prompt use of amyl nitrite, but in general the remedy is useless, and it is doubtful if anything is gained by such attempts.

During an attack little can be accomplished. The head should be protected, a cork or piece of wood inserted in the mouth to protect the tongue, and, later, when vomiting occurs, the patient should be rolled on the side to avoid a possible suffocation or suction pneumonia.

Of the various drugs in use it is to be insisted upon that they are purely palliative. With the advantages go serious disadvantages. The most efficacious are the bromid preparations. The bromin ion acts as a motor depressant, both on the cortex and spinal cord. It may, therefore, simply put a damper upon disordered cortical functions. It does not alter those functions materially. It may seriously be questioned whether bromids are really not more harmful than helpful. They certainly will prove to be disastrous if given without discrimination and in the routine fashion so frequently followed.

They are, therefore, a last resort, or an emergency brake when attacks mount up in increasing numbers and severity in status epilepticus.

Mixtures of the bromids of sodium, ammonium, and potassium are best given, preferably, in large doses of water.

The amounts are to be determined by the frequency of the attacks. These should be carefully plotted. If occurring with any regular periodicity the drug is best started a few days before the expected onset, run up in fairly good doses, and then abandoned. It is folly to dose a patient day in and day out for attacks occurring bi-monthly or even monthly.

In status, or threatened status, the bromids are well combined with opium, chloral, veronal, or other motor depressants.

Bromids have little value in the psychical equivalents. They are sometimes useful in quieting the ~~excessive~~ irritability of these patients, but in general other remedies are much more valuable.

Toulouse and Richer have introduced a modified bromid therapy by eliminating common salt, NaCl, from the dietary, and introducing NaBr in soups, in breads, etc. Certain observers have reported good results, others negative results, and it is uncertain if the ideas of Toulouse and Richer have proved fruitful or not. The restriction of intake of sodium ions may play a part in the general value of the treatment, and if Ca ions are added the results are thought to be much better. All of this, however, is much in the air at the present time. One thing is certain, however, and that is that the reduction of chlorin ions works disastrously in furthering bromid intoxication, hence, if NaCl is to be left out, $CaCl_2$ should be added, or other chlorid, not sodium or potassium.

Bromid preparations have sprung up in great numbers in the last few decades. It cannot be said that any have special advantages. Certain patients do better with one, and others with another, and whether it is to be strontium bromid, bromocol, bromopin, bromolin, etc., will depend upon individual elimination tests.

In all bromid therapy it must not be overlooked that bromid salts are stored up in the body, and that bromin retention with chlorin excretion readily brings about bromid poisoning. Thus when the body fluids become poor in chlorin, and the heart and kidney functions are not active, bromid intoxications appear as general apathy, and dulness, or delirium.

It may be seen from the foregoing that cardiac and renal medication should go hand in hand with bromid therapy. Digitalis, belladonna, chloral, arsenic, water should be utilized freely.

The opium bromid therapy of Flechsig has not borne out the expectations of its founder.

Other remedies suggested have been borax, amylene hydrate, chloretone, zinc, urethane, solanum carolinense, simulo, trional, and veronal. None are specifics, all may be helpful at times.

From our present viewpoint concerning their multiple causation a serum treatment of the epilepsies, as a general procedure, is nonsense. It is a remnant of the era when the loose concept of autointoxication seemed to explain everything, but in reality explained almost nothing.

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CHAPTER XIX

LOCALIZED MUSCULAR SPASMS

SIDNEY I. SCHWAB

Under the term localized muscular spasm are included all those voluntary, semi-voluntary, or involuntary movements of muscles or muscle groups which have for the most part no definite purpose, no conceivable part in the conservation or protection of the individual, with the exception of the tic movements, which almost always in the beginning are definite, or, rather, might be interpreted as definite attempts at defense or safeguarding the individual. Such movements are the expression of hypermotility of the nervous mechanism and, as far as we can see at present, are in no way essential for the preservation of the fundamental physiological laws underlying muscle movements.

As a class these movements are little understood etiologically, although their clinical manifestations are by now matters of such frequent experience that their division into distinct types is amply justified.

In treating such conditions, with the causal factors so doubtful and the anatomical substrata for the most part so undefined, we are forced to be contented—that is, with the exception of one or two types—with efforts of a very general nature. These are chiefly aimed to correct the manifest end products of the disease and an attempt at the general treatment of the whole organism, with the hope that some of the phenomena of the disease may be due, perhaps in an indirect way, to a general underlying cause. In some of this group it should not be forgotten that there is a very distinct psychical involvement. This is particularly true of the occupation neuroses and the tics. In these types there is open a field of treatment in which very definite attempts at education and muscle training should have a distinct and important place.

It might be said in this brief introduction that in none of the diseases considered in this group is there at the present time any question of specific treatment; that is, in none of these diseased types is there one universally used method, either chemical or otherwise, which can be considered as having a direct and uniformly positive effect upon the causative agents of the malady; therefore, treatment is largely experimental and, in a sense, personal.

In outlining methods of treatment, then, only those means will be described which either by experience have proven effective, or which by logical consideration meet as many factors as is possible in the etiology and consequent symptomatic expression in the way of complete clinical pictures.

TIC

Tic, or tic movements, may be defined as the resulting motor phenomena remaining as a permanent effect in a muscle group or groups concerned in the beginning in the production of a peripheral intended and well coördinated movement or group of movements. The origin of such a tic movement or movements is usually protective and aimed at conserving the individual.

As a variant to such movements are those which have become habitual, or the results of imitation and suggestion. There is likewise a group of movements frequently difficult to differentiate from the tic movements which seem to be the remnants of choreiform attacks. The dividing line between a group of tic movements and a choreiform movement is extremely indefinite, particularly so when the sensory origin of the tic movement is not to be obtained in the history of the case. In a general way there are two chief indications to be met as far as the treatment of tic is concerned. The first has to do with the discovery of its origin—frequently, as has been before suggested, psychical, or, at any rate, sensory; and, second, methods which aim at the production of educational exercises and rest.

The type of a tic movement which is the result of the sensory stimulus is that form of facial spasm which is known as tic convulsif. This condition consists of spasmodic and contractive movements of the facial muscles as a result of an attack of intense neuralgia of the fifth nerve. It is quite obvious that the tic movement itself is not subject to treatment, and that efforts must be made to correct the originating factor of the movement, which is pain localized in the distribution of the fifth nerve. With the subsidence of the neuralgic pain, that part of the phenomena which is included under the tic movement group disappears.

This form of facial contraction as a result of pain in the fifth nerve and as an accompaniment of the preëxisting neuralgia of the trigeminus is usually known as tic douloureux, and treatment must be directed toward the cause of the tic movement.

The primary spasmodic movement of the muscles supplied by the seventh nerve, known as facial spasm, is comparatively common. This consists of spasmodic, fibrillary tremor of the muscles of the lips, cheek, eyelids, and, in fact, all the muscles supplied by the seventh.

The facial spasms, or convulsive tic, must be sharply distinguished from a true tic limited to the face. The general distinguishing points are chiefly found in the character of the movement itself, which is localized in each muscle, and to the failure of the psychogenic origin in the case of spasm. In addition, a true tic movement is seldom limited to the distribution of one motor nerve.

In regard to the treatment of facial spasm naturally a great deal must depend upon the severity of the case and the discomfort it causes the patient. The attacks show a great variety in their periodic frequency. Long periods of time elapse when the patient is not troubled at all; at other times the spasm becomes very troublesome, so much so that the patient is in a constant condition of discomfort.

There is no specific remedy in a medical sense for this condition. Naturally, all etiological causes must be sought for removal, if removable, and treated, if that is possible. Patients who have this disorder are not, as a rule, of a nervous type, and seem to demand very little in the way of general treatment. The spasm being strictly local in character, and limited absolutely to the distribution of the seventh nerve, treatment, to be effective, must be entirely local in character. An indication to be met in this instance, as in other cases of excessive and useless muscular movement, is rest. If it is possible to obtain a cessation of movement within the spasmodic territory for some length of time, there is a chance that the spasm may cease altogether. It is needless to mention that drugs, as such, cannot accomplish this end. It has been suggested, therefore, that a temporary paralysis of the seventh nerve at the point of its exit at the stylomastoid foramen would effect the result intended, by paralysis of this group of muscles. The nerve has been injected with alcohol at this place and paralysis of the muscles supplied by the facial has resulted and the spasm, naturally, has been stopped. Inasmuch as it takes the facial nerve at this point from 8 months to 2 years to regenerate, during that period the patient is relieved of all symptoms. There has been scarcely enough experience with this method of treatment to indicate anything of its value, and it naturally is only in very desperate cases that it should be recommended. While the operation itself cannot be considered dangerous, yet the results of paralysis of the seventh nerve can never be accurately determined. There is a possibility that with the return of function to the seventh there may be considerable asymmetry of the face on that side.

For the milder cases there is little to be done except, possibly, attempts at educational control and efforts made to distract the patient's attention from the group of muscles that are affected. It is a common experience in these cases that worry, attention, and concentration directed toward the facial nerve affected seem to increase the number and severity of the attacks. Naturally, measures planned to minimize these factors would be of service. Especially to be avoided are emotional explosions of various sorts and fatigue produced by too much speaking, singing, chewing, etc.

There seems to be a very marked relationship between the fatigue and spasm, and, naturally, the most logical method of treatment would be directed toward minimizing this possible cause in the production, or increase, of the spasm.

A special form of facial spasm might be here mentioned, that known by the name of blepharospasm. In these conditions the spasm seems to be limited to the orbicularis of both eyes. It is questionable in some cases just how far these should be called localized muscular spasms and how far they are but a part of a tic movement. At any rate, whatever their origin may be, treatment should be largely directed toward the correction of defects in vision and the correction of any irritating process in the eye itself.

Spasm limited to the motor branch of the trigeminus, or the so-called masticatory spasm, is of seldom occurrence, but when it does occur there is found a tonic, or clonic, movement of the masseters in which the mouth is opened and shut rapidly as in the motions of chewing.

Spasm in the muscles supplied by the hypoglossal, or so-called glossal spasm, and in the muscles supplied by the glossopharyngeus, are conditions which are not found of themselves but are generally a part of some form of a neurosis, frequently a symptom of hysteria. Their treatment rightfully belongs to that of the underlying conditions.

Spasmodic movements in the various parts of the body form clinical pictures which are sufficiently differentiated from one another to merit separate types and classifications. The gross underlying etiology of these conditions may be very difficult, and, therefore, therapeutic indications are largely met in the study of the causes which are productive of the clinical pictures. In Oppenheim's classification the muscle-group spasms are divided into those of the neck, those of the trunk, and those of the extremities, and this classification may be adapted here, looking toward clearness in their therapeutic consideration.

A spasm, or spasmodic movement, of the muscles of the neck may be included under the term accessorius spasm, and is generally found in the muscles supplied by this nerve. They are sometimes one-sided and sometimes double-sided. Although the beginning of the spasmodic movement may be somewhat limited to the muscles supplied by one nerve or confined to a muscle group having the same general function, yet with the increase and persistence of the movement it tends to involve other muscles, even those far reaching. In this way a spasmodic movement confined at first to the sternocleidomastoid may soon spread until practically all the muscles of the neck are involved, and the movements thus produced are very complicated and tortuous.

The clinical picture presented by spasmodic movements of the neck muscles is too well known to need character description. It is obvious that the therapeutic indication is best met if the source and distribution of the movement are first accurately determined. First of all, it is very essential to differentiate a spasmodic movement from a tic movement confined to the same group of muscles, because the therapeutic problem would be very different in one case from the other. It might be added here that the differentiation is sometimes very difficult, if not impossible, especially when the movement due to spasm of the muscles becomes more or less stereotyped and constant. When the differentiation is made and it is found that the case is really one of spasm of a group or groups of the neck muscles the first indication is to discover, if possible, whether the source of irritation may exist as a preliminary starting point for a sensory impulse starting the spasm into action. The irritating process may be located anywhere within the sensory range of the section overlying the muscles involved in activating the movement; particularly the painful points over the cervical vertebræ, in the glandular supply of the neck, middle ear, teeth, and vertebral bodies must be investigated. In rare instances removal, for the alleviation of the sensitive areas, has been of great benefit.

In regard to the purely medical treatment it has frequently been found that patients suffering from spasmodic movements of the neck muscles are of the neurotic type, and therefore measures planned to correct by educational means and very hygienic measures such defects, whether congenital or acquired, are to be recommended. It is questionable whether there is any place for a purely medicinal treatment for this affection; that is, by the use of drugs, as such. As has been stated before we possess no chemical combination which has any direct effect upon the excessive action of muscle fiber, and there is no means at present available of applying a drug of this kind directly to the muscles involved.

Electricity has been tried very frequently, either in stimulating the group of muscles involved in the movement or in stimulating an antagonistic group, in the hope that a muscle balance might thereby be established. Considerable experience has shown, however, that in the long run such methods are not successful.

Exercises planned to train antagonistic groups are likewise to be recommended in certain cases, and much can be accomplished by attempts at developing movements in antagonistic groups of muscles. The same criticism that has been applied to exercises in the exercise treatment of tic movement is here advanced; namely, that the attention and interest of the patient are directed too much toward the malady and that thereby the intensity and the extent of the movements are increased.

There is always a possibility in spasmodic movements of the neck muscles that resort to surgical measures might offer some therapeutic advantage, and, while the number of cases that have thus been treated is not large, the underlying basis upon which the surgical procedures are based seems sound enough to warrant their being tried in a few cases. It might be here stated that one of the chief objections to the employment of surgical measures is that the total extent of the muscular spasm is difficult to obtain, and unless the whole group of muscles affected is treated the operation will probably be a failure.

The muscle-cutting operation by which the muscle groups affected are severed and the head placed in an exaggerated opposing position has not been productive of great success, chiefly because when the muscles become reunited the movement is reëstablished in its original intensity.

Section of the nerve supplying the muscles has likewise been attempted, but in such cases the danger of a permanent paralysis with atrophy of muscle groups is an objection.

Theoretically the most logical form of surgical treatment that has yet been suggested would be that which has been designated as muscle-group isolation. In this procedure the nerves supplying the groups of muscles that are affected by the spasmodic movement are temporarily paralyzed by injecting their nerve with alcohol, and during the period in which the nerve thus paralyzed becomes degenerated the opposing muscle groups are exercised in order to overcome the spasm as it develops. No case in which this procedure has been effective is as yet known to the author, but the suggestion is here advanced that it seems capable of some utility.

Tic movements are the result of some sensory stimulus which is responded to by a simple or a combined movement. The tic movement remains after the sensory stimulus has disappeared or been forgotten. In this sense it is seen that the tic movement is largely psychogenic in its origin and is in the nature of a defense movement. The variety of such movements is very great; they may be from a simple movement, such as blinking the eyelids and complicated movements of the arm or leg, and, in fact, of the whole body. There is a certain impulsive feature in most complicated tic movements which show any evidence of persistency. This impulsive feature is quite a part of the tic movement itself and lies more chiefly imbedded in the patient's consciousness than the motor element. Just what the nature of this impulse is is not quite understood but can be observed in the careful study of many different types of tic movement that the patient experiences, not only the impelling desire to perform the movement but, associated with that, a feeling of relief after the movement is over. In fact, it is a common experience that patients with tic movements seem to feel rather better after the series of movements is completed than in their absence.

In any therapeutic endeavor directed toward the cure or betterment of tic movement the psychical element should never be forgotten, for it is chiefly by a careful consideration and study of this feature of the disease that successful therapy may be obtained.

For the simpler tic movement there are several methods which might be tried. First of all it is essential, as in the localized spasms, to determine if there is any sort of peripheral irritation which in any way might be connected with the performance of the movement. If any is found, naturally it must be removed, if possible, or altered. Following this it is essential to study carefully the muscle groups which are involved, and, if possible, to obtain either from the history of the case or by other means the origin and the course of subsequent development of the movement from its earliest beginning. It is very well at this stage to explain to the patient, if he is sufficiently intelligent and mature, just what a tic movement consists of, what its origin is, and what methods should be best employed for its removal. The simpler movement can, therefore, be best treated by exercises which have for their purpose two principal features, one of which is the artificially induced control of the movement itself, and, secondly, the control or delay of reflex movements which are located in the group of muscles which are affected.

In addition to all of this, generalized treatment directed toward establishing in the patient artificial control and various automatic actions should be instituted. For example, exercises in slow and deep breathing, exercises directed toward the establishment of the voluntary control over the patient's general movements during succeedingly increasing periods of time. In this way and by this simple means there is laid the foundation for exercise of volitional mastery of the movement; that is, as far as it is possible in the individual case.

In order to control a simple tic movement the patient is directed to look into a mirror and note the number of times the tic movement in the face is produced, then he is directed, by steadily gazing into the same mirror, to reduce the number of movements in any given time by one-third or one-half, as the case may be. These exercises should be graded very carefully, so that the patient is not fatigued nor irritated nor discouraged; more than five minutes three times a day is hardly to be advised, and the exercises should be kept up for a number of weeks or months.

The exercises directed toward the voluntary control of ordinary reflex movements are accomplished in the following way: The patient is directed to exercise his volition in studying ordinary reflex acts of a protective kind. For example, he is directed to keep his eyes open when the movement is made toward the eyeball, which ordinarily would result in the protective closing of the eyelids. In this way the patient accomplishes a slowing up, as it were, of the reflex activity of that particular group of muscles.

While such exercises are sometimes very effective in the simpler kinds of tic localized chiefly in the muscles of the face, they fail utterly quite frequently in the more exaggerated tic movements in which one or more extremities of the body are involved. In such cases we are face to face with a very complicated problem. After we have exhausted all the ordinary means of treating such movements; that is, by rest, various sorts of antagonistic movements, and other things referred to above, there is evidently nothing left but a deeper study of the origin of the tic movement, with methods that require a peculiarly specialized training. Reference here is particularly made to the study of Janet, Freud (v. Vol. I, Chapter XI, Section I, and others concerning the subconscious origin of many abnormal movements which must be included under the general term of tic. It is obviously impossible in a treatise of this sort to more than mention the methods that might be used. The psychoanalysis of Freud in the hands of especially trained students can be used effectively for the study in regard to the complete origin of certain groups of tic movements, the theory being that if a deeper origin of such a movement is once obtained and the patient becomes consciously aware of it the movement itself will disappear or be repressed.

A certain number of tic movements are certainly to be regarded as a motor expression of certain abnormal mental activities, the nature of which is not known to the patient, and the origin of which may be found in long-forgotten infantile emotional experiences. Just what types of tic could be included in this class cannot be altogether ascertained with certainty, but it is evident, however, that the neurological therapeutist in cases of this kind must exhaust every possible endeavor to obtain relief for his patient, and the psychoanalysis of Freud and the hypnotic methods of Jana and Prince should be attempted if other things fail.

A further method of treating certain complicated tic movements where the movement results from successive activating muscle groups is artificially induced rest, either of the whole extremity involved or of a segment of the extremity. The best means of inducing such a state of complete rest is by paralyzing the peripheral nerves. This can be done by various methods of which, perhaps, the alcoholic injection in the sheath of the nerves seems the most advantageous. The principle involved in this method is that a paralysis extending over many months or, perhaps, two or three years would involve a complete loss of the motor memory of the movement, and thus, with the slow regeneration of the nerves that have been injected, the tic movement would lose its power of reproduction owing to its motor amnesia. So few cases have been treated by the latter method that the suggestion is merely made in this place that it is possibly a seemingly well-founded therapeutic procedure.

Paramyoclonus multiplex was described first by Friedreich in 1881. It consists of a clonic, jerking movement chiefly found in the muscles of the extremities and the trunk. The movements are short, lightning-like, and are very similar to the reactions obtained by a galvanic automatic current. The disease is rather rare, and is sometimes found associated with epileptic attacks. Therapeutically an endeavor must be made to differentiate the diagnosis of paramyoclonous from hysterically produced muscle movements. In the latter case, naturally, treatment should be only indirectly directed toward the abnormal muscular movements.

There is no definite treatment effective in paramyoclonus. Electricity, bromids, chloral, and other sedative drugs have been tried, but the results have not been satisfactory, nor does any drug medication which has been attempted seem to check the progress of the disease. Still, in any case a method of treatment should be devised which includes a possibility of attacking the epileptic nature of paramyoclonus, and probably a carefully regulated régime in regard to diet, exercise, and rest, with a moderate amount of bromids, would show the best results.

OCCUPATION NEUROSES

Under the term occupation neuroses have been included from time to time conditions of local overuse of muscles in various trades and occupations, practically all of these conditions having the same basic factors, etiologically considered; that is, fatigue produced by the overuse of muscle groups or the use of muscle groups in constrained or unnatural positions causes what may be considered localized spasms. The term occupation neuroses has undergone, however, since its inception a variety of interpretations. By some they are considered as a part of the great questions of occupation under the term industrial diseases, and the factors chiefly at work are indirect ones, such as long hours, bad conditions of work, worry, and other extrinsic factors which have nothing positive to do with the clinical side of the case.

There is running throughout most of the cases of occupation neuroses a general condition to which the term neurasthenia has been given, meaning by that an easily and pathologically produced fatigue and pathological conditions of nervous irritability. If this basic condition exists, then it is easy to understand how the overuse of a particular group of muscles in the performance of some definite business should show itself by weakness, tremor, spasm, etc. The rôle of alcohol, tobacco, and other habit conditions plays a somewhat important part as an indirect cause in the production of these conditions, for it is frequently found, particularly in the male subjects of occupation neuroses, that a period of alcoholic indulgence preceded the outbreak of the neurosis. While this is true in many cases, it cannot be accepted as a positive fact in all. The therapeutic importance of this rather broad view of the occupation neuroses is essential, for adequately planned treatment should include all the possible factors which are at work. Whatever the cause may be and whatever the type is, the general lines of treatment may be laid down as applicable in practically all cases. Inasmuch as overuse and fatigue must be considered as direct factors, the chief indication of treatment is to meet these conditions. This is done chiefly by means of absolute rest: A rest directed not only to the group of muscles that are involved, but rest in the larger sense; that is, rest to the individual, which means in ideal conditions, naturally, and a total cessation from work of any sort is a matter of practical importance. However, it is usually found that those who are the subjects of occupation neuroses are not in a position to take a vacation, but are dependent absolutely upon their trade. In such cases the therapeutic problem is a difficult one and the physician is forced to be satisfied with half-way measures. The next therapeutic principle, if absolute rest cannot be obtained, is then its nearest competitor; that is, a change of employment, by which other groups of muscles are used, would be advised. For example, in the case of a so-called writer's cramp or telegrapher's cramp the individual should change his work for the time being and employ himself in such tasks, if possible, as include no particular group of muscles in their activity. This can in some cases be carried out with a fair degree of comfort in a material way to the patient.

The other factors that have been mentioned before should be zealously gone into. The use of alcohol, over-indulgence in habits of various kinds, improvement in the hygiene of the individual, discovery of all sources of worry and their removal, or change, if possible, and everything that would be considered of importance in a general condition of easily produced fatigue of the nervous system should be corrected. It is only upon a basis of this kind that success can be met with in the treatment of the local conditions, and the patient should be so informed.

In regard to the treatment of the muscles involved there are many things that are of advantage; that is, after the general basis has been investigated. It is of prime importance that the patient himself should be carefully instructed as to the causation of his trouble, and his co-operation could be thus obtained and his understanding could thus be made clear. After a total rest for the muscle groups that are affected, the treatment that has proved the most successful is very slowly graduated exercises, never approaching the point of fatigue, of the muscles which are chiefly concerned. It has been found of considerable advantage to perform these muscle movements while the hand or arm is under water as hot as can conveniently be borne. The purpose of this maneuver is to remove as far as possible the effort necessary in the performance of such movements. It is surprising to see how effectively the patient can carry out a complicated muscle group movement with the hand fully immersed than otherwise would be the case. Very light massage and electrical excitation of the muscle group are sometimes likewise effective.

In a general way, if a foundation is laid for the treatment of the individual as outlined above and then treatment directed to the muscle group is followed afterward, a fairly good result should be obtained in the majority of cases. If, added to this, there can be substituted an employment which does not necessitate the continued use of a single muscle group, and if the patient can possibly be brought into a state of satisfaction with his new employment, and, further, if the economic conditions incident to the new employment are as favorable as, or more favorable than, the old ones, a perfect result can be obtained. It is seldom, however, that these ideal conditions can be met with. The physician, as a rule, has to take what measures he can, considering the special features of the individual case.

It is well, again, to accentuate how much more important it is to treat occupation neuroses from the very broadest standpoint and regard them very much as we regard industrial diseases; that is, the causes producing them are allied with general occupational problems, and the individual, as a rule, is the unconscious victim of these conditions.

CHOREA MINOR

Chorea minor, or, as we term it at present, infectious chorea, is a disease which is caused by some infectious process closely allied to endocarditis, rheumatism, tonsillitis, etc. The choreiform movements which are chiefly concerned here are an expression, from the standpoint of the nervous system, of this infection. The treatment, however, of this condition can be disassociated from the treatment of the general process underlying the causation of such movements. As a rule the treatment in a case of acute infectious chorea is simple and satisfactory. Sometimes a more severe type is met with, where the therapeutic problem presents considerable difficulties. This type should be considered by itself, not because it is different etiologically, in all probability, but because it presents special problems of its own. The treatment of acute cases of infectious chorea is easily summarized. The condition being due to an acute infection the patient should be regarded as being distinctly sick, and rest in bed and quiet, almost to the point of isolation, are the prime requisites for successfully treating the case. Secondly, being due to an infection, and in this sense being self-limited, an acute attack shows a distinct tendency to recovery no matter what is done. There being no specific drug for this disease, as there is no specific drug for any of its allied conditions, therapeutics in a drug sense are largely of secondary importance. Owing to its being closely related to rheumatic infection, such medication as is found to be successful in these conditions is found equally successful in chorea; that is, the salicylates, and particularly aspirin in small doses, are the prime elements to be relied upon. Rest in bed, quiet, isolation, small doses of aspirin therefore form the basis of the therapeutic endeavor. The choreiform movements themselves are generally greatly improved by these simple measures—in fact, rest alone will frequently cause them to practically disappear.

It need scarcely be mentioned here that the heart requires particular attention. In a large percentage of chorea cases a slight affection of the heart is shown frequently by slight enlargement, by an increase of the pulmonic second sound, and at times by a systolic murmur. This condition, however, soon disappears and in uncomplicated cases needs no further attention.

Restlessness and insomnia in some cases of chorea can be best treated by hydrotherapeutic measures. After the first week of the disease warm baths before the child is put to bed have a remarkable effect, and it is not uncommon to see the child rest comfortably and easily as a result. A good routine to follow in cases of the first attack of chorea in comparatively young children is absolute rest in bed, preferably out of doors if possible, warm baths at night before sleep, and a diet largely of milk, total isolation, and, if necessary, screening of the bed if the patient is in a ward of a hospital. This, in addition to small doses of aspirin, in the early days of the onset of the disease accomplishes, as a rule, very positive results. [According to my experience, arsenic is the most valuable remedy in chorea minor. It matters not in what form it is prescribed, provided it is given in slowly but regularly increased doses until symptoms of mild intoxication develop. When this occurs gradual reduction in dosage is ordered.

In those cases in which violence, intensity, and continuance of the movements threaten life the paramount indication is to control these movements. For this purpose chloral is given in ascendingly large doses, which are very well borne by children, until the contractions have stopped for twelve hours (Bouchut). When any untoward symptoms develop, the remedy must be withdrawn. In many cases the disease is cured by this method, in all cases it is mitigated.—Editor.]

It must not be forgotten that, for reasons at present not understood, the first attack of chorea seems to predispose the child to a second attack and a third attack. These attacks seem to have a seasonal occurrence, particularly toward the spring of the year. The second attacks, as a rule, are less severe than the earlier attacks, but there is some danger, if the attacks are repeated, of their becoming more or less chronic; that is, the patient never being absolutely free from certain manifestations of chorea movements. This is important for the reason that there is frequently found in the more chronic forms of chorea a close analogy in symptoms to tics of various sorts, and as the methods of treatment are very different in these two conditions a differential diagnosis should always be made if possible. It is, however, frequently a matter which cannot be determined with any accuracy, the tic movements and the chorea movements in their impure types being absolutely without specific characteristics.

There are a number of other forms of chorea which must be noticed from the point of view of treatment. The chorea associated with pregnancy is well known. In this form the chorea is never a disease of itself, but simply a complication, and bears no distinct relation to the acute infectious chorea we have just been considering. There is no treatment necessary for the chorea in pregnancy other than rest and quiet, the chorea movement as a rule disappearing completely after the pregnancy is over.

The degenerative form of chorea, the so-called Huntington chorea, is largely a family disease. In this type the chorea movements are accompanied by a progressive mental deterioration. The disease is absolutely incurable and is progressively fatal. Of course, the treatment in such cases would have to be largely prophylactic, based upon the consideration of eugenics, and would scarcely come into the realm of treatment in the ordinary sense.

There are forms of movements in childhood of a bizarre type, conforming to no special clinical picture found in typical chorea and having a variety of causes. The name of chorea electrica was given to these curious movements. The name is not a particularly happy one, as the symptoms have nothing whatever to do with electricity, either as a causative factor or a descriptive one. Most of these curious movements to which the term chorea electrica has been given are tic-like movements of a complicated sort, which either spring from or are the objective expression of a subconscious experience. In childhood, naturally, an investigation directed toward the discovery of the origin of these movements would be the proper therapeutic method to pursue. It is sometimes questionable whether a child should be submitted to such an analysis, and therefore all other methods should be tried before this is attempted. It is only in rarer cases that it is necessary to submit the child either to the Freudian psychoanalysis or investigations following the Janis method in hypnotic conditions.

The imitative choreas, or the so-called habit choreas, which are sometimes imitative in origin and sometimes, apparently, are not, should be treated from a broad point of view of educational training in children. This would imply, naturally, physical methods and other general measures directed toward the establishment of automatic control of muscular movements.

There is a form of congenital chorea movement which is exciting a great deal of interest although the disease is rather rare. In this condition the chorea movements date from birth or early infancy and never disappear completely. There is evidently in such cases a congenital insufficiency of muscular inhibition as far as the intended purpose of the movements is concerned. The cases that have been described have been found, as a rule, in otherwise normal individuals. In these cases the physical examinations showed absolutely nothing abnormal. Naturally, any effort made toward modifying conditions such as these must be planned to establish, even if only a little can be accomplished in this way, the automatic inhibitory control which is lacking. Exercises must be devised to establish this control, at first in a voluntary way, and then by constant repetition to cause the voluntary attention to become more or less automatic.

There are several other movements included under the type of chorea which are found as isolated phenomena, as complications of various sorts of conditions, such as chorea movements found in hemiplegias, athetosis, in organic diseases of the brain, chorea movements found in hysterical affections, the chorea movements sometimes following severe emotional shocks, etc. All of these should be treated not from the point of view of the movements themselves, but from the standpoint of the patient himself.

PARALYSIS AGITANS

Paralysis agitans, from our present knowledge of the disease, must be considered one of unknown origin affecting the nervous system largely from the muscular point of view, producing typical tremor, rigidity of the musculature, paresthesia of various kinds, associated frequently with changes in the osseous system of an arthritic kind, particularly in the spinal column. This leads frequently to changes in gait and posture, which further intensify the clinical picture presented.

Paralysis agitans was formerly considered to be the result of senile, or pre-senile, changes in the central nervous system, depending chiefly upon the early development of arteriosclerosis, but with the study of paralysis agitans cases in comparatively young children in whom there was no trace whatever of an early arteriosclerosis this theory has seemed somewhat untenable. From the therapeutic point of view the origin of the disease, or the theories that have been advanced to explain the disease, are not of importance.

The task of the therapeutist in treating paralysis agitans is directed to the following considerations: First, the disturbance in motility, as shown chiefly by the tremor and rigidity; second, the paresthesias, and third, the mental state of such individuals.

The amount of discomfort in cases of paralysis agitans can be very great. Such patients are sometimes never comfortable in any one position, the muscle rigidity preventing them from relaxing sufficiently to adopt comfortable attitudes. For that reason patients with paralysis agitans have frequently great difficulty in sleeping, it being impossible to retain a relaxed position sufficiently long in bed to allow them to fall asleep.

The tremor is likewise a very disturbing factor, not only from the esthetic point of view, but because the patient cannot use his hands or arms with any definite purpose for a long-continued period of time. In this way hand workers, or those whose employment necessitates manual dexterity of any sort, are rendered incapable of pursuing their tasks.

The paresthesia, feeling of stiffness and strangeness in the muscles are often torturing symptoms.

It can be easily seen, therefore, that the disease presents a problem sufficiently broad to interest a therapeutic endeavor to its utmost.

As a rule most of the paralysis agitans cases that are not of the senile type show very little variation from the normal in the general physical condition; in fact, a great many are very vigorous and healthy individuals, so that the disease offers no chance at all for general treatment, there being nothing to correct.

Naturally, in the aged, or in those in whom there are found some complicated conditions, a consideration therapeutically of such conditions should be given. There is no specific therapy for paralysis agitans, and, like all diseases which have no definite treatment, the variety of methods which have been suggested are very numerous.

For the tremor, which in so many cases is the prime symptom presented by the patient, there is little that can be done in the way of specific treatment. We have no drug that will control the muscular movements of a tremor type and, at the same time, not affect the general muscular strain. Of all drugs that have been suggested hyoscin hydrobromate is the only one that offers any encouragement, and that in only a comparatively small percentage of cases. It is particularly unfortunate that in arteriosclerotics, in which class most of the Parkinson types belong, this is a dangerous drug. Its usefulness in paralysis agitans depends upon long-continued use and progressively increasing doses, and until a sufficient amount has been given to affect the tremor in some positive way the doses are to be increased. Naturally, in individuals over 60 years of age such methods of medication are open to serious question.

For the muscle rigidity nothing seems so efficacious at present as bathing and massage, the patient obtaining very often great relief from alternating baths and gentle massage before going to bed. Electricity of any kind has only a very temporary utility. After all, what can be done is mostly in the way of intelligently handling the individual and using such means as are suggested from time to time which tend to increase the patient's comfort. A moderately active life, with the interests of the patient kept artificially alive, if necessary, is of far greater value than temporary improvement obtained by this or that drug therapy.

V. TROPICAL DISEASES

CHAPTER I

INTRODUCTION—THE RÔLE OF THE MEDICAL MAN IN THE FUTURE CONTROL OF THE TROPICS

William B. Wherry

Encircling the earth, between 30° N. and 30° S. of the equator, are tropical and subtropical regions—the most beautiful, the most fertile, the most richly endowed portions of the globe. Time and again they have been invaded by northern races in search of wealth. Stricken by strange pestilences of mysterious origin, the invaders disappeared. Gradually the rumor spread and the belief became ingrained that there lay "the white man's grave." One hundred years ago Dr. Jas. Johnson wrote: "Whoever has attentively examined the posterity of DeGama, and Albuquerque, now scattered over the coast of Malabar, the plains of Bengal, and the island of Macao, once the theaters of Lusitanian preëminence, will be tempted to exclaim:

> " 'Twas not the sires of such as these
> Who dared the elements and pathless seas;
> Who made proud Asian monarchs feel
> How weak their gold was against Europe's steel,
> But beings of *another mold,*—
> Rough, hardy, vigorous, manly, bold!"

Will racial decadence be the fate of those whites who are now contemplating an expansion into the tropics in the near future? Will modern Science, operating through the medical man, be able to neutralize the forces which act deleteriously upon the white man in the tropics? Upon the solution of these questions must rest the white man's future relation to the tropics.

Leaving out of consideration the results of the old spirit of discovery, conquest, and religious zeal, the effects of which upon the present ownership of the tropics are scarcely perceptible, there is one factor above all others which has instigated the seizure of tropical possessions by modern white races. With the overcrowding of the temperate zones and ultimate scarcity of food ever before them, these peoples have sought to provide room for expansion. That the tropics already have been largely appropriated may be seen from the following table, prepared for Sir Wm. Osler by Mr. H. O. Becket of the Department of Geography, Oxford, England. It shows at a glance the tropical possessions of the four western nations —England, France, Germany, and the United States—in 1909.

	Tropical Territory In				Total Tropical	Home Country
	America	Africa	Asia	Pacific		
France	**440,000**	**17,700,000**	**18,500,000**	**80,000**	**36,720,000**	**39,000,000**
	35,000	4,032,000	310,000	9,000	4,386,000	207,000
German Empire	Nil.	**11,700,000**	Nil.	**400,000**	**12,100,000**	**60,000,000**
		931,500		96,000	1,027,500	209,000
United Kingdom	**2,000,000**	**30,500,000**	**296,600,000**	**1,150,000**	**330,250,000**	**44,500,000**
	109,000	1,600,000	1,900,000	1,400,000	5,009,000	121,000
United States	**305,000**	Nil.	Nil.	**7,707,000**	**8,012,000**	**76,000,000**
	47,500			134,500	182,000	2,970,000

Heavy figures give population. Lighter figures give area (in square miles).

However, it is extremely uncertain that these possessions will serve for the purpose of expansion. With characteristic audacity the white man is attempting to accomplish in the near future that which has resulted from the operation of natural laws acting through thousands of years—the acclimatization of an alien race. It is true that the problems facing white settlement in the tropics a comparatively few years ago have been greatly modified by recent advances in our knowledge of tropical diseases, but these researches tend rather to promote the success of the temporary settlement of white labor in the tropics and to increase the efficiency and supply of the black labor of the tropics than to guarantee their successful and permanent settlement by whites.

Formerly it was held that heat, the use of peculiar foods, the abuse of alcohol, sexual excesses, the lack or abuse of exercise, etc., were the factors which lowered the resistance of the white exotic transplanted to an unaccustomed environment. Recent researches have convinced many that most of the damage was due to protozoon parasites—the Entamœbæ, the Plasmodidæ, the Leishmania, the Trypanosomidæ, and metazoon parasites, such as Uncinaria, Clonorchis endemicus, Opisthorchis noverca, Schistosoma japonicum, Porocephalus armillatus, etc. It is quite possible for hygiene to protect the white man from these dangers; but, granted that this has been accomplished, can he be insured immunity from the injurious effects of tropical sunlight?

According to present belief, the white man to become climatically naturalized must accomplish two things: he must acquire more pigment in his skin and go for the greater part unclothed, and, in addition, readjust his thermoregulatory mechanism—nervous and cutaneous. This can never be accomplished excepting through the operation of the slow and uncertain laws of natural selection.

Those who lay special stress on the significance of the pigment in hides and hairs of geographically distributed animals call attention to the fact that the pigment in the skin of all races is the same, and the amount present in each is at least sufficient to exclude the dangerous rays of sunlight encountered in the particular latitude of the particular race home. Hence, the blackest skins are found in unforested Africa, the plains of India; yellowish and brownish skins in jungle tribes or on the humid coastlands in the tropics, or in the desiccating grass lands in intemperate climates; the whitest skins in high latitudes with moist atmosphere and cloudy skies. Similarly, among the lower animals are cited, as extreme examples of adaptation, the zebra and the polar bear.

However, there seems to be no proof that the actinic rays in tropical sunlight exert more than a superficial effect on man. It has not even been demonstrated that the sunlight contains rays of sufficient penetrating power to reach the pigment deposited in the deeper layers of the skin. Nor is there proof that it contains a greater proportion of ultraviolet rays; there is only proof, to date, of its greater intensity. Freer and Bacon have shown that the chemical activity of the sun's rays is from 5-20 times greater in Manila in October, November, and February than in Chicago during the months of May and June. They encountered great variations in its activity, e. g., two successive, equally clear days were actinic and non-actinic respectively. The destructive action of sunlight on many microorganisms is established, and it is known that plants cannot survive geographic changes without the elaboration of sufficient chlorophyll of the type necessary to absorb light of particular wave lengths, but it does not follow that these arguments can be applied unhesitatingly to the action of light upon higher organisms. Yet, on the basis that pigment is developed according to need, it has been advised that the white man settling in the tropics must dress in clothing which is opaque to light or of a color sufficiently near the lower end of the spectrum to exclude the dangerous rays. The results of such protection have been conflicting and generally unsatisfactory; but this question needs to be reinvestigated, for, according to Freer, recent work shows that color is immaterial as long as it presents a considerable absorption band in the proper region of the spectrum.

The other side of the argument, namely, that the injurious effects of the tropics on whites is due to the intense heat combined with excessive moisture, seems to have been more definitely established. Recently Aron has brought forward very convincing experiments showing that the heat-producing rays exert a more injurious effect than the actinic upon animals naturally provided with a heat-regulating mechanism. The rapid, deadly effect of tropical sunlight on certain mammals could be obviated by measures which promoted loss of body heat, e. g., loss through the action of an electric fan. He further showed that brown skins, which should theoretically absorb more heat in sunlight, show less rise in temperature in tropical sunlight than the skins of whites under similar conditions. This difference is attributed, and it would seem correctly so, to the earlier and more rapid loss of heat by water of evaporation. So that the main difference, in their relative resistance to a tropical climate, between naturalized and alien races lie chiefly, if not entirely, in the delicacy and efficiency with which cutaneous heat regulation is performed. Evidently those measures promoting heat loss, e. g., going uncovered as much as possible, protection from sunlight, frequent bathing, the liberal consumption of cooling, non-alcoholic drinks, etc., are only secondary in importance to the prevention and eradication of infectious diseases. In short, as Johnson's motto expresses it, we must "study well the clime, mold to its manners our obsequious frames, and mitigate those ills we cannot shun."

However, putting aside for the present the question whether the white man can permanently acclimate himself in the tropics, the fact remains that the tropics are largely in his possession, and that he will have to face the problem of developing their enormous natural wealth as a source of supply for an overcrowded world—not by the old reprehensible system of exploitation, but by holding and developing them as a trust for civilization.

It is amazing to what extent the English-speaking world is already dependent upon the production of the tropics. Benjamin Kidd gives the following instructive figures:

1896—United Kingdom trade with the tropics..	£138,000,000
1895—United States trade with the tropics.....	70,000,000
Total..........................	£208,000,000
Combined trade of the United Kingdom and United States with the remainder of the world outside of English-speaking lands...........	473,000,000

The importance of the tropics to the English-speaking world as a whole is further made apparent when one considers that the trade of the United Kingdom and that of the United States with the tropics in 1896 and 1895 respectively amounted to some 38 per cent. and 65 per cent., respectively, of their total trade with the remainder of the world.

It is not likely that the inhabitants of the temperate zones will grow less dependent on imports from the tropics, unless scientific agriculture develops a system of intensive cultivation undreamed of at present. Noone believes that the natural resources of the tropics will be developed by native races—as witness Brazil and Central America. An adequate supply can be obtained only by the use of temporary white labor or by native labor under the supervision of a temporary white population. The world-wide efforts which are being made to make the tropics habitable for whites will insure the success of temporary settlements of white labor. These have been shown to be economical in Manila, in the Panama canal zone, in the Queensland sugar plantations, etc. Further, by removing the parasitic factors in negro lethargy, they will increase the efficiency of black labor; by decreasing mortality they will increase the supply of this black labor. So that the study of tropical diseases is not only of scientific but also of economic interest.

For further details concerning hygienic victories in the tropics, the works of the late Sir Rubert Boyce and that of Sir Ronald Ross are especially recommended; and for details concerning the influences of geographic environment the reader is referred to the researches of Ellen Churchill Semple.

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CHAPTER II

INSOLATION

PAUL G. WOOLLEY

Insolation (siriasis, sunstroke, heat stroke, thermic fever) is a term applied to certain symptom-complexes that are the result of disturbances of heat regulation. These disturbances arise primarily from physical causes—the heat and water content of the air about the body, the condition of the peripheral vascular circulation, etc.—but these causes lead to or produce chemical changes in the organism which are manifested in the characteristic clinical symptoms.

According to the sequence of the symptoms, that is, according to their rapidity of development and their severity, various classifications have been made in the group of insolation. The simplest is that which includes heat exhaustion and sunstroke. The former refers to syncope from exhaustion "caused either by the direct rays of the sun or a heated atmosphere in the shade." The latter refers to the results of "the general heating of the whole body, blood, and tissues, which may happen either from prolonged exposure to the sun's rays in a heated atmosphere, to a heated atmosphere in the shade, or to an abnormal thermotaxic state due to vasomotor or other heat-regulating disturbances."

From this statement it appears that there is little justification for a classification other than a much simpler one which would arrange the cases into mild, severe, and peracute. The causes of all are obviously the same. The symptoms are phases of the effects of heat upon bodies which differ in one or another direction or degree. The type and amount of variation is determined by many factors—the use of alcohol, overeating, overwork and fatigue, bad ventilation, anxiety, worry, personal uncleanliness, wearing of too tight or too heavy clothes, and others.

An epitome of this argument is found in a recent article to the effect that heat exhaustion is produced by high temperature; heat exhaustion brought on by exposure to the sun is sunstroke!

Insolation has always been attributed to exposure to the sunlight, and more particularly to exposure of the head to the direct rays. While this is true, in part, it is not true of all cases, and it does not account at all for the large group of border-line cases that are classed as heat exhaustion or prostration, which are encountered among stokers, laundry workers, and persons who are crowded together or who live in hot, badly ventilated, insanitary rooms, buildings, or barracks. Taking the whole series of cases that may be included in the group (represented by the classical hyperpyrexial sunstroke, by atypical apyrexial heat stroke, and by heat prostrations which occur in the absence of direct sunlight), we may assert that insolation may occur under any circumstances in which heat accumulates in the body, and in which the body does not rid itself of its metabolic end products, notably perhaps acids. And, since metabolism increases under the influence of abnormal heat, it will appear that symptoms of insolation are imminent whenever the heat production of the body exceeds the heat dissipation.

Hirsch (12) believed that, while heat was the primary factor, the immediate cause of trouble was a diminution of oxygen and retention of toxic principles. Vincent (17) also stated his belief in a toxic basis. Gordon (10) says that recent work tends to the conception of the paralyzing action on the nervous system of some toxic element which produces metabolic changes in the neurons, and, according to the degree of autointoxication, the effect of the sunstroke will be either an attack of ordinary heat prostration or syncope with unconsciousness or death. If this be true, then insolation is an autointoxication brought about by substances formed in the body under abnormal conditions of heat retention.

How the bodily heat regulation is upset we do not know, but, from the fact that heat accelerates chemical reaction, and from the fact that the symptoms of sunstroke are also largely the symptoms of shock, we may surmise that there is some chemical point common to the two conditions. The only probable common factor that occurs to one is the increased acid content of the tissues. In sunstroke this may be the result of increased katabolic chemical activity under the influence of heat. However this may be, we are told that the alkalinity of the blood is decreased and that the oxygen content of the body is diminished.

It is, however, with the problem of solar rays in their relation to insolation that workers have busied themselves, and it is work upon these problems that has given most insight into the question of the production of insolation.

For a time men were led to suppose that the violet and ultraviolet part of the spectrum was most active. This supposition was based upon the results of experiments on the effect of ultraviolet rays upon bacteria, protozoa, and plants, which showed beyond question that these lower forms of life were seriously affected by these rays. It has, however, been shown that the results reached with the organisms that have no heat-regulating apparatus do not apply to the other organisms that have such an apparatus. Also, Freer (8), Gibbs (9), and Bacon (2) have shown that the tropical sunlight (Manila) contains no more ultraviolet rays than does the light of temperate zones (Chicago). And yet in tropical climates heat prostrations are more numerous (in troops especially) than in temperate climates. Experiments with underwear which prevents the action of ultraviolet rays on the body have also been unproductive of good results. More interesting still, however, are observations such as those of Chamberlain (3), which render it very doubtful that the actinic rays play an important part in tropical deterioration of any sort.

In the absence of any direct evidence of the ill effects of the ultraviolet rays, the attention of Aron (1) was turned to the red and infrared rays, with the result that very definite and useful information has been developed.

The heat rays may be active in one or two ways, or in both. They may act by increasing the temperature of the air, or by heating the body itself. Either action tends to decrease the heat dissipation from the body, or to increase the heat accumulation in the body. Aron's work indicates that both ways are important.

The body loses heat in three ways—by conduction, by radiation, and by evaporation. Under normal circumstances radiation is the most active, but, as the surrounding temperature rises, radiation becomes necessarily less important and evaporation (perspiration) becomes more active, until at temperatures which equal or exceed that of the body evaporation alone is of value. Vierordt (16) estimated the normal heat losses through various channels as follows: the urine and feces, 1.8 per cent.; by expired air, 3.5 per cent.; by vaporization of water from the lungs, 27.2 per cent.; by evaporation from the skin, 14.2 per cent.; by radiation and conduction, 73.0 per cent. Rubner showed that the heat losses, estimated in calories, by radiation and conduction, took place according to the following table:

Temperature	Calories Lost by Radiation	Calories Lost by Evaporation
7° C.	78.5	7.9
15° C.	55.3	7.7
20° C.	45.3	10.6
25° C.	41.0	13.2
30° C.	33.2	23.0

These figures indicate that under normal circumstances radiation is the most important method of physical heat regulation, but that under abnormal circumstances evaporation becomes the most important. Without increased evaporation the body would not merely retain its heat, but would absorb heat from its surroundings. This state of things had led Hill (11) to remark that, so long as evaporation is active, high temperatures alone will not change the temperature of the body. It is only when evaporation is insufficient that heat produces serious results.

This is shown in a remarkably clear way by Aron's experiments with dogs, cats, guinea-pigs, and rabbits, in all of which evaporation takes place only through the respiratory tract. Aron shows first of all that dead animals, in which there is no possibility of heat regulation of any sort, absorb heat very rapidly when exposed to heat, either in the shade or in the direct sunlight, and that heat accumulates far more rapidly in the sunlight than in the shade. He shows, also, that, when in living animals heat dissipation is diminished by reducing the possibility of evaporation (by tracheotomy), the temperature of the animals rises much more rapidly than in normal animals, but less rapidly than in dead animals, while at the same time control animals, which have also been tracheotomized, but which are kept under otherwise normal conditions, live comfortably and show no abnormal variation of temperature and no symptoms due to heat retention.

In a series of experiments with monkeys Aron showed that these animals, which are as accustomed to tropical climate as any that can be obtained, when exposed to the direct sunlight, quickly died, though the control animals kept in the shade, even that of an umbrella, survived. But he also showed that monkeys kept in the sun, but in a current of air, such as that produced by an electric fan or a natural breeze, suffered no damage. He further showed that, if monkeys were placed in an apparatus so that their heads were exposed to direct sunlight, but with their bodies protected and kept cool, they suffered no damage. He concludes, therefore, that it is not the direct effects of the sunlight which do damage, but the hyperthermia produced by the heat rays. He believes that the heat radiated from the sun warms the body tissue more rapidly than can be compensated for by the regulatory mechanism of the body. The tissues and the blood increase in temperature to a point higher than is compatible with life. Apparently the organs most susceptible to this increased heat effect are the brain and heart, though it is not indicated from the lesions in these organs which are most essential in causing death. These characteristic lesions are hemorrhagic lesions in the brain and (in monkeys) in the heart.

Not less clear than the results of his experiments with animals are Aron's results with human beings. They indicate, as he says, "that the temperature of the human skin increases in the sun, but does not reach the normal body temperature. In animals without sweat glands the skin temperature rises above febrile heights, and the tissues lying underneath are heated. On the other hand, penetrating of heat through the human skin seems improbable, the effect of the rays being neutralized by water evaporation on the skin. The more perfect this water evaporation is the better the normal body temperature may be maintained." "Monkeys exposed to the sun in Manila die in little over an hour because of their limited capacity to evaporate water, while man, with his well-developed sweat glands, resists the same climatic condition for a much longer period without detriment." The great importance of this evaporation is shown by the remarkable fact that a man lying quietly in the sun lost 280 grams in weight in one hour. The above figure does not include the water held in the man's clothes, which was also actually lost to the body. The volume of respired air in such a man increased 23 per cent.

The foregoing facts account very well for the usual cases of sunstroke. There remains, however, a small group of cases in which but slight increase of temperature have been observed. Aron accounts for these upon the basis of sudden loss of large quantities of water. He quotes P. Schmidt, who observed, under unfavorable climatic conditions during muscular work, water losses as great as 1 kilogram per hour. He proceeds to call attention to the fact that, since the body cannot withstand a loss of water of more than 10 per cent. of its weight, an acute loss of 3-4 kilos must necessarily result in grave disturbances, collapse, or death. In this connection it is to be remembered that large losses of water may occur with little reduction of the body temperature, as, for instance, when the sweat runs down and off the body "in streams." Where losses of water are so great, evaporation is reduced, and refrigeration decreased. Serious results may, therefore, result from paralysis of the vasodilators on the one hand, or by too great loss of water from the sweat glands on the other. "It is not the sweat we see, but that which we do *not see,* which exerts the cooling effect."

One further point of interest (among many others) is taken from Aron's work, and this has to do with the question of the exposure of the human head to heat. Aron states that the temperature of the air in the thick black hair of a Filipino may reach 50.1° in an hour of exposure.

PROPHYLAXIS

If the foregoing facts are true, then prophylaxis consists in preventing the accumulation of abnormal amounts of heat in the body. To accomplish this there are mediate and immediate methods. The former applies to habits and clothing—in general to hygienic surroundings at any time; the latter to habits during severe hot weather.

To enable oneself to withstand great heat the regulatory system of the body should be kept in the normal state of adaptability to circumstances. The skin should be kept clean to allow free evaporation; the clothes should be loose and light, moderately absorbent, but thin enough to allow the air to circulate freely, and so assist evaporation. The diet should be one that will not create unnecessary demands upon the system. Alcoholic drinks should be tabooed, plenty of cool water or fruit juices may be indulged in to advantage, since they serve to keep the excretory system freely open and to encourage refrigeration by evaporation. Fatigue, worry, and anxiety should be avoided. Heavy work in the open should not be done, and even in the shade should be limited. Dwelling houses and living rooms should be open, the windows shaded in such a way as to exclude the heat rays without cutting off what breezes blow. Electric fans or punkahs are often indispensable, and cool (not cold) baths are infinitely refreshing as well as useful in keeping the body at ease.

During times of intense heat, and always during the hot hours of the day, all physical effort should be reduced to an absolute minimum. At such times rest in the shade, with as little clothing as circumstances permit, and, if possible, where even the slightest breezes blow, should be freely indulged in. Under such circumstances electric fans, when possible, and punkahs, where they are not, are not only pleasant, but almost necessary. The oriental custom of taking a siesta in the middle of the day is a highly commendable one which Europeans should be counseled to follow. In addition to these peaceful recommendations, it is advisable to suggest a light diet accompanied by as much water as can be gratefully consumed. Such a régime will result in quiet of mind and body and will encourage a useful, moderate degree of perspiration and evaporation, which will result in a comparatively mild state of refrigeration. Attention should be paid to the excretory organs. Citrous drinks encourage the renal activities, and so assist in the cleansing of the tissues. Much water will assist intestinal elimination and so assist in preventing intoxication from fecal stasis. Constipation must be avoided with as great care as diarrhea. Any exposure to the direct sunlight should be avoided as much as possible, and when such exposure is necessary it should be brief. An umbrella is a valuable protection.

TREATMENT

The treatment of insolation is rather an unsatisfactory matter. This must be so because of the grave changes that are produced in the various organs of the body by the action of the intense heat and the products of abnormally rapid (and very probably aberrant) metabolism. One cannot expect to remove extravasations of blood in the brain, meninges, or heart in a few minutes or hours, and still less can one expect to restore the damage produced by the hemorrhages in less than a very considerable time. Moreover, the disease is rapid. Its victims fall where there are no therapeutic resources. They must be carried to some place where remedies are at hand; where ice can be had, where baths can be given.

There are two main objects in view in treating any form of insolation: to reduce the heat content of the body and bring about elimination of its toxic contents. When these things have been done, *tempus edax rerum* alone will attend to the hemorrhages, the destruction of tissue, the fatty degeneration, and the rest. But even time will not always allow the body to return to its former state. Often residual symptoms remain, such as a tendency to relapses, to headache, to partial loss of memory, to loss of sustained attention, to polyuria, or even to glycosuria. There is the further possibility that insolation, particularly the moderately severe type, predisposes to meningitis.

Treatment of an attack of insolation is, therefore, one that combines refrigeration with elimination, both active enough to produce good effects without embarrassing organs, such as the heart, which are already damaged. To accomplish the former the practice is to give ice cold packs or baths. To compass the latter, recourse is commonly had to stimulants. This seems not to be the best, except as a last resort. Stimulation of an already burdened organ can do little good and much damage. Hydrotherapeutic methods are of more value in insolation than any drugs. Ice packs or iced baths have a great value, but should not be continued for more than a few minutes, 1-2 (Chandler, 5) at a time, and should be discontinued when the rectal temperature has reached 104° F. To replace the water lost to the body before the attack, and to increase elimination, there is no better method than infusion of saline solutions. If it be true that the oxygen content of the body is low and the acid content high, then such solutions should be alkaline. Such alkaline solutions as those recommended by Fischer (7) are extremely efficacious, whether given by rectum or intravenously, in neutralizing the acids of the body and increasing water elimination by the kidneys. The solution for rectal use should be prepared as follows:

Sodium chlorid	10 grams
Sodium carbonate ($Na_2CO_3.10\ H_2O$)......	10 grams
Water	1,000 c. c.

The injection should be given slowly enough to allow retention. The time consumed in injecting a liter should not be less than one hour.

For intravenous injection the following solution may be used:[1]

Sodium chlorid	14 grams
Sodium carbonate (crystallized)............	10 grams
Water	1,000 c. c.

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[1] For intravenous use the alkaline saline solution should be very carefully prepared. The carbonate cannot be boiled. The salt solution should therefore be made and sterilized, after which the crystals of carbonate may be added. *If the crystallized sodium carbonate is not available and use must be made of the ordinary dried form of the salt, the amount indicated in the above prescription should be divided by three.*

This also should be given very slowly. The effect of these solutions upon the secretion of urine is remarkable, and, as a rule, they will make it unnecessary to use digitalis. When this latter drug is used, it should be very carefully administered and its effects carefully watched. The use of strychnia is not advised in the active stage of the disease.

Such treatment will dispose of the immediate danger, and when this has been done treatment is symptomatic. Headache may be treated with acetanilid, phenacetin, salicylates. But these drugs should be used with extreme caution. This is particularly true of the coal-tar products. Sleeplessness may be controlled with bromids, trional, or other mild sedatives and soporifics.

In apyrexial heat exhaustion external hydrotherapeutic measures are uncalled for, and treatment should be eliminative and stimulant. The internal hydrotherapeutic methods should be very useful in these cases and should be combined with friction, massage, warm packs with sufficient internal stimulant medication.

After recovery from an attack of insolation great caution must be observed by the patient to prevent recurrences from subsequent exposures to heat. He must pay careful attention to prophylactic measures, and use the greatest precautions in ordering his life. It is advisable for all such persons to arrange their time in such a way that they may spend the hot seasons at some high altitude or cool summer resort. Many tropical countries have what is known as a "summer capital," to which many people go to escape the heat of the lowlands.

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CHAPTER III

AFFECTIONS DUE TO POISONOUS REPTILES, FISH, PLANTS, AND ARTHROPODS (INCLUDING MYIASIS)

CREIGHTON WELLMAN

In this section, and also in the following sections written by me, I have not hesitated to include brief discussions of several little known ailments. Frequently the most puzzling problems of physicians in the tropics are unusual and sometimes comparatively slight ailments, rather than the classical and carefully studied diseases described in so many textbooks. The list is, however, not complete on account of space considerations.

1. OPHIDISM

Poisonous snakes may be said to be of two kinds, Colubrine and Viperine. To the former group may be referred the venomous Hydrophine water snakes. Among tropical Colubrines may be mentioned the corraline snakes (*Elaps*), the cobras (*Naja*), the kraits (*Bungarus*), the Australian black snake (*Pseudechis*), the tiger snake (*Hoplocephalus*), the death adder (*Acantophis*), the bush master (*Lachesis*), and the sea snakes (*Hydrus,* etc.). Some of the important Viperines are the ketten (*Daboia*), the puff adder (*Clotho*), the rattlesnakes (*Crotalus*), the moccasins (*Triconocephalus*), and the copperhead (*Denisonia*).

It is often impossible to ascertain the species or genus of snake that has inflicted a given bite, and, while Colubrine and Viperine venoms act somewhat differently, the treatment of their effects can best be discussed together.

There appear to be hemolytic, leukolytic, hemorrhagic, and neurotoxic bodies and coagulins included in snake venoms, and the proportions and effects of these seem to be different in different kinds of venom.

My own observations have been made principally with *Clotho arietans,* one of the most dangerous Viperines known, and which is common in southwest Africa. The principal effects of its bite, as studied in my cases, are as follows: loss of the coagulability of the blood after passing through the tissues at the site of the bite, extravasation of blood from the mucous membranes, including the stomach and nose, the remote selective action of some ingredient of the venom noticed by its effect on the respiration, sloughing of the site of the puncture, and the frequent precipitation of active attacks of malaria in patients who survive the direct effects of the bite. The bite of the house cobra (*Naja nigricollis*) observed by me in the same region, gives rise to effects much less severe, although the hemolytic power of the venom appeared to be greater *in vitro* than that of *Clotho,* which latter observation agrees with those of Lamb on Indian snakes.

The general conditions to be combated in cases of snake bite are shock, increasing pain at the point of puncture, local edema, hemorrhage, sloughing, vomiting, small, frequent, irregular pulse, dyspnea, difficult breathing, convulsions, coma, and death. Ecchymoses due to a destructive action of the venom on the endothelial cells of the capillaries, fatty degeneration of the parenchymatous organs, and necroses of the liver occur.

REMEDIAL MEASURES

These should aim at the neutralization of the venom absorbed by the tissues, at the prevention of avoidable absorption, and at the relief of dangerous or distressing symptoms.

Specific Treatment.—Calmette's antivenin should be promptly administered in all cases of snake bite occurring in the tropics, because it lessens the degree of hemolysis and appears to inhibit the effect of the neurotoxin present. At least 100 c. c. should be injected, preferably intravenously, as promptly as possible after the bite has been received. Subcutaneous injection into the neighborhood of the bite is also advised in addition, but the serum should not be introduced in the exact location of, or together with, the permanganate solution referred to below.

Other Treatment.—Ligation of the bitten limb above the wound, excision, sucking, cautery, etc., are not to be neglected. Hot iron, nitric acid, or other caustic may be used for this last.

From my experience, I am personally strongly in favor of injecting a weak solution of permanganate of potassium into the tissues at the site of the wound.

In addition to the above, various observers have advocated different measures, among which may be mentioned transfusion of blood or salt solution, bleeding at the same time from the bitten limb. Subcutaneous injections of a 10 per cent. solution of calcium hypochlorite (10 minims) have also been mentioned, as well as pilocarpin hypodermically. Chlorid of gold has likewise been advocated.

For local edema applications of ammonia water have been used, and to combat the symptoms of collapse various heart stimulants, galvanism, rubbing of the limbs, coffee, ammonia inhalations, strychnin, and alcohol (brandy or whiskey given with care to avoid overdosing). Promptness in treatment is the greatest factor in the prevention of a fatal issue.

Prophylactic Measures

Among prophylactic measures may be mentioned snake killing, stimulated by rewards; the cultivation of land, and the keeping of reptilivorous animals, such as the mongoose. Crude carbolic acid smeared around the supporting posts or other foundation of houses will keep out house-loving species.

2. OTHER POISONOUS VERTEBRATES

A. Batrachia and Lacertilia

Among the Batrachia may be mentioned the salamanders (e. g., *Cryptobranchus japonicus*), and the toads (e. g., *Bufo agua*). Of the Lacertilia there is only one important poisonous genus, viz., *Heloderma* (*H. horridum*) from tropical America.

Salamandrin and the poison of *Heloderma* are said to act much like viper venom. Several toxic substances have been studied from toads: bufotalin, a heart poison; bufotenin, a nerve poison, and phrynolysin, an erythrolytic substance. Here may be mentioned bufagin, from the so-called parotid glands of *Bufo agua,* identical or closely allied to epinephrin.

Remedial Measures

In the absence of specific antisera the poisons of the above are best combated by the general measures given for snake bite.

B. Poisonous Fish

There are many species of tropical fish which are protected by the possession of spines, which are provided with a poisonous substance furnished by glands situated at their base, e. g., the cat fishes (Siluridæ). Many other genera are similarly provided. Among these may be mentioned the sting rays (Trygonidæ), species of *Pelor, Scorpæna, Trachinus, Synaceia,* etc. Still other fish (*Muræna, Stomias, Tetradon,* etc.) cause trouble by their bites.

The conditions set up by the bites and stings of such fish are chiefly local, i. e., inflammation and edema accompanied by much pain, fever, lymphangitis, lymphadenitis, and occasionally gangrene. The general manifestations are nausea, vomiting, purging, cold sweats, sometimes delirium, and even convulsions.

Further, a number of tropical fish, not always easy to distinguish from edible species, are the cause of disagreeable and not seldom grave symptoms when eaten as food. These fish are found throughout most of the tropical seas, and cases of poisoning frequently occur, especially in sailors, marines, and newly arrived residents. Some of the most important fish of this character are certain herrings (*Clupea*), parrot fishes (*Scarus*), sea porcupines (*Tetradon, Diodon*), cat fish (Siluridæ), barracuda (*Sphyræna*), file fish (*Balistes*), trunk fish (*Ostracion*), lip fish (*Lachnolemus*), and a few Atherinidæ (*Tetragonurus*). Some varieties (e. g., *Caranx* and *Thynnus*) are only occasionally poisonous, being, as a rule, found edible. I have seen a species of *Barbus* in West Africa cause alarming symptoms when eaten.

The picture is uncommonly like that of ptomain poisoning. Burning of the fauces, nausea, headache, fainting, collapse, and death from paralysis of the heart and respiration have been recorded. Paralysis or gastritis may follow as sequelæ. The poison of *Tetradon,* which has been most carefully studied, is said to contain a base (tetradin) and an acid (tetradonic acid). The mortality from *Tetradon* poisoning is stated to be over 68 per cent.

Remedial Measures

Stings or bites may be treated according to the general procedures given under snake bite. After the eating of poisonous fish emetics or the stomach pump are to be used at the onset of the first symptoms. Stimulation is called for, and strychnia hypodermically has specially been recommended. Artificial respiration may be indicated.

Preventive Measures

Great care should be taken to identify all tropical fish before eating. Among the natives some individuals are immune to the ill effects of eating these fish, so caution should be displayed in accepting the assurance of guides, etc., as to the harmlessness of any certain variety of fish.

C. POISONOUS MAMMALS

The Australian *Ornithorhynchus paradoxus,* by means of poisoned spurs, may inflict bad wounds. The poison is stated to be much like snake venom in its action, although much milder.

The rat bite disease of Japan is characterized by a dark eruption, fever, and a tendency to recur.

The bites of carnivora are to be considered as open, infected wounds.

Remedial Measures

The treatment of *Ornithorhynchus* bites consists of the general measures used in cases of snake bite. No special treatment is recommended for rat bite disease. The ill effects following the bites of carnivorous animals may require treatment for general septic infection or even rabies.

In my own experiences with bites of large cats (leopards, lions, etc.) the measures called for were purely surgical.

3. BITING AND STINGING ARTHROPODS

These belong to the centipedes, spiders, mites, ticks, and various families of insects. The species are too numerous to be detailed here. Possibly infection introduced at the time of the bite is to blame for the symptoms in some cases, rather than any venomous substance. The effects of the bites and stings of Arthropods in the tropics are often quite severe, and may even cause death, especially in children and in adults with heart affections. The Durango scorpion is stated to cause the death of 250 persons each year. The conditions set up by the various poisonous species of arthropods are very similar and are usually marked by local edema and inflammation, pain, occasionally sloughing of the wound, nausea, vomiting, thirst, nervous manifestations, a weak, rapid pulse, dizziness, dyspnea, and sometimes convulsions and death. In cases of scorpion sting observed by me in West Africa I found agonizing pain at the point of puncture a marked feature. When the puncture is on the mouth or tongue suffocation may take place from swelling.

Remedial Measures

If the wound is on a limb, *ligate* above the puncture, and the wound should be incised, sucked, and cleansed. For the swelling a compress of ammonia is said to be effective. I have found that *hot applications* are effective in relieving the intense pain sometimes following the puncture of scorpions.

Cocain has also been recommended for relief of the pain. Sedatives which do not depress the heart may be administered in addition. Calmette's antivenin is reported to be effective against scorpion venom as well as snake bites. The serum prepared by Todd in Egypt may prove to be valuable against scorpion stings.

4. STINGING AND IRRITATING LARVÆ AND PLANTS

The larvæ of many species of tropical Lepidoptera are covered with hairs or spines which possess strongly poisonous properties. In Africa I found the families Limacodidæ, Arctiidæ, and Liparidæ very troublesome. One patient was even forced to return to Europe because of frequent and distressing urticaria arising from this source. Many similar offenders from other parts of the tropics have been reported. Coleopterous larvæ also cause similar effects. In this connection may be mentioned two species of African Millipedes (*Spirostreptus* and *Odontopyge*) which I found to be highly poisonous. Many poisonous species occur in other parts of the tropics.

Here should be mentioned also the terrible itching and burning set up when the poisonous hairs or juice, by means of which certain tropical plants defend themselves from being eaten, come in contact with the human body.

Remedial Measures

Wash with an alkaline solution and afterward smear with vaselin impregnated with camphor and menthol.

If blisters occur and break, treat antiseptically. Any sores that ensue should be carefully treated, as the inflamed skin shows a decided tendency to ulceration.

Preventive Measures

Care regarding sleeping on the ground and under trees should be observed. Native reports regarding stinging plants are usually reliable. All the African stinging Malvaceæ have lobed leaves and can thus be recognized.

5. MYIASIS

The invasion of the skin, natural orifices, deeper tissues, and digestive tract of man by dipterous larvæ is often productive of grave destructive processes and even death. In the tropics many such processes are met.

A. Cutaneous Myiasis

This condition is principally caused by two species of flies. The first of these is an African form, belongs to the Muscidæ, and its larva is variously known as Ver du Cayor, Tumbu, Inyo, etc. Gedoelst considers that there are four different species which breed in the skin, but Grünberg classes all the larvæ under one species, *Cordylobia anthropophaga.* The larva of the South American fly is called, in different regions, Ver macaque, moyoquil, torcel, berne, nuche, gusano, ura, macaw fly, etc. The species is *Dermatobia cyaniventris* and belongs to the family Œstridæ. Some authors consider that the fly affecting man in certain parts of South America is a different species (*D. noxialis*). At any rate, the two flies are closely allied. A similar fly occurs in Tonquin and another in tropical Africa. Another species, also a Muscid, which may be mentioned here, is *Auchmeromyia luteola.* The larva, variously known as Ovaenyo, the floor maggot, etc., sucks blood from mammals as do ticks or bugs. Its normal food is human blood.

The Ver du Cayor is found only in the tissues of man or animals and is a subcutaneous parasite. Monkeys, rats, and various other animals are affected. The larvæ ensconce themselves in the tissues, as warble flies do in cattle. I have often squeezed these maggots, which give rise to boil-like formations, from the skin of natives and domestic animals. They seem specially to affect babies and dogs, and more than three hundred larvæ have been reported from a single puppy, and as high as sixty from a single child. The ovipositor of the female is incapable of piercing the skin, and the young larva must bore its own way into the tissues. It is not known if the parent lays eggs or living maggots. The danger to the victim is from septic or infectious material being introduced by the grub. The presence of the larva is self-limiting, since it emerges as an adult in about a week. The Ver macaque is also normally a subcutaneous parasite of mammals, and attacks man in the same manner. Unlike the foregoing, however, the larval stage of this fly lasts for many months. In addition to man, pigs, dogs, cattle, agouti, jaguar, mules, and birds are invaded. The fly is sylvan in its habits and especially affects domestic cattle. While the bite of the African floor maggot is in itself insignificant, yet the abstraction of blood is considerable, and the danger of septic or infectious material being introduced is not negligible. On one occasion I allowed larvæ bred from captive flies to bite my forearm in several places, and they produced wheals which were the seat of marked burning and itching, which did not entirely disappear for four days. In native kraals the larvæ are most abundant under sleeping mats, where children who urinate during sleep have lain, showing that the adults or larvæ, or both, seek such places as promising sooner or later the presence of suitable blood-possessing hosts. The gravid females seek dark corners, where they sit motionless till ready to oviposit.

Remedial Measures

In the case of the Ver du Cayor and the Ver macaque the maggots should be squeezed from the skin by firm pressure and the cavities cleansed with an antiseptic solution and protected from subsequent infection. For bites from the floor maggot antiseptic and soothing applications are indicated. Mentholated vaselin was the most effective in allaying itching and smarting in my own case.

Preventive Measures

In order to avoid the Ver du Cayor and Ver macaque the body should be kept covered while in endemic areas. Houses should be screened, and proximity to domestic animals should be avoided, especially in forest countries. The larvæ should be destroyed whenever found. In order to escape the attacks of the floor maggot cleanliness and avoidance of sleeping in native kraals are recommended. Old huts should be burned. Screening of dwellings is indicated. The adult flies are fond of fermented matter and may be trapped in numbers by using vinegar as a bait.

B. Myiasis of the Natural Orifices of the Body and of Wounds

The most important of the flies with these larval habits is *Chrysomyia macellaria,* belonging to the family Muscidæ. This dangerous fly is found from the Argentine Republic to the southern United States, and has the habit of laying its egg masses in wounds or in the ears and nostrils of persons sleeping in the open. It has also been found in the mouth and vagina. The fly likewise attacks wounded animals, and has even been found in dead bodies. *Chrysomyia viridula* has habits similar to the preceding, and has been reported in wounds, from British Guiana, and in the nasal cavities, from the West Indies. Over a hundred maggots were discharged from the nose in one case. Other similar cases caused by species of *Pycnosoma* have been reported from India under the name of Peenash. In one case about one hundred larvæ were removed from the nose and sinuses by irrigations, and the patient recovered. The flesh flies (Sarcophagidæ), which are viviparous, also produce destructive myiasis. In tropical Africa I produced destructive nasal myiasis in goats, using both *Sarcophaga africa* and *S. albofasciata. Sarcophaga chrysostoma* is also reported from British Guiana as attacking wounds. Probably *Sarcophaga ruficornis* is also the subject of some accounts of Peenash. Various undescribed species of *Sarcophaga* have also been reported in this connection. Patterson reports terrible and fatal nasal myiasis by flies of this genus. Hundreds of maggots were removed from the patient. These flies also attack cattle, cats, and dogs. Harrison also reports fatal facial myiasis in British Honduras, in which about three hundred maggots belonging to the genus *Sarcophaga* were removed. Many other cases are reported.

Remedial Measures

The larvæ are best killed by injections of chloroform, benzin, turpentine, carbolic solution (2 per cent.), or some other substance which will destroy the maggots without injuring the tissues too severely.

If necessary, the frontal sinuses, the antrum of Highmore, and other bony cavities should be opened by trephining or chiseling. After the larvæ are killed they should be carefully removed and the cavities cleansed and irrigated with some antiseptic solution.

Preventive Measures

Sleeping in the open in tropical countries, especially by any one with ozena or fetid breath from any other cause, should be considered dangerous. Uncovered wounds are also liable to invasion by these flies. A rational prophylaxis would consist in keeping all cooked food carefully covered, and in careful use of a mosquito curtain during the *siesta*. It should also be pointed out that in endemic regions care must be taken to avoid allowing flies to oviposit on the anus during the act of defecation.

C. INTESTINAL MYIASIS

There are several reports of the presence of fly larvæ in the intestinal canal of patients in the tropics. The flies usually belong to the genera *Fannia* and *Sarcophaga*. The writer reported the first case of intestinal myiasis due to *Fannia desjardensi*.

The presence of the maggots gives rise to abdominal pain, bloody diarrhea, and other dysenteric symptoms. Thickly coated tongue, foul breath, abdominal tenderness, and nervous symptoms were also noticed by me. The prognosis, as a rule, is good.

Remedial Measures

In my own cases I employed santonin and castor oil as in the case of infection with intestinal worms. The result was prompt and complete.

Pumpkin seeds followed by a purge have been recommended in similar instances. Probably the purge without a vermifuge would be effective.

Preventive Measures

My cases were probably infected through eating food which had been left uncovered after cooking. A rational prophylaxis would consist in keeping all cooked food protected from flies. The danger of exposure of the anus has already been pointed out in a preceding paragraph.

D. CHIGGERS AND AINHUM

Closely related to the foregoing conditions are the habits of *Sarcopsylla penetrans,* the chigger, chigoe, ewundu, or sand flea, to mention but a few of its common names. This parasite receives, as a rule, but slight attention at the hands of writers on tropical diseases. The animal, however, merits more consideration than it generally receives, as it not seldom plays a significant rôle in the production of mutilation and disease. The insect is a common pest which, originally from South America, has invaded and entirely traversed Africa, entered India, has even been reported from Cochin China, and is evidently on its way around the world.

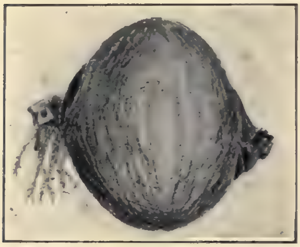

FIG. 1.—FEMALE PREGNANT CHIGGER (*Sarcopsylla penetrans*). Magnified. (After Wellman.)

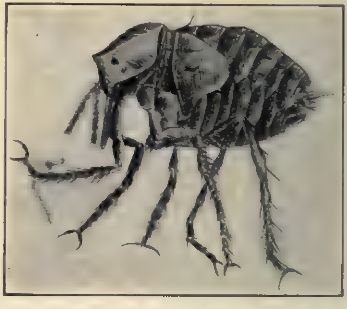

FIG. 2.—MALE CHIGGER (*Sarcopsylla penetrans*). Magnified. (After Wellman.)

It usually produces no serious lesions, yet I have seen shocking deformities, loss of limbs, gangrene, septicemia, and death result directly or indirectly from its presence. *Apropos* of its being the possible transmitter of specific microorganisms, I may mention that I have found various bacteria, including *Bacillus lepræ,* in chiggers, but the insect has not been carefully studied to determine if it be a true carrier of such germs. With reference to the disease called ainhum, esola, etc., I may say that in West Africa I noticed that the chigger has, even in healthy feet, a predilection for the under surface of the little toe, and especially so if, as is often the case with barefooted peoples, the skin is cut or torn. It is well known to those who have studied the insect that it invades abraded or irritated surfaces oftener than sound skin. The fold under the proximal joint of the little toe corresponding to the web between the toes is the point where wounds are oftenest made by the sharp grasses, etc., through which the native walks and runs. A principal reason for this is that the little toe of barefooted blacks especially lies separate from the others at an angle due to anatomical reasons connected with flatfootedness. When wounds are made here the chiggers persistently invade them and must be removed constantly. So these insects seem to play a part, in many instances at least, in the continued irritation which, especially in blacks who have a fibrogenetic tendency, leads to the contracting fibroses resulting in ainhum.

Remedial Measures

Chiggers should be promptly removed with a clean dull knife whenever they are noticed under the skin. In case the tissues are injured in the process, a bactericide should be applied. All infected lesions should be laid open and thoroughly cleansed. It is occasionally necessary to remove toe nails in order to do this.

When ainhum is advanced amputation of the offending toe is all that can be done. In beginning cases it has been suggested that the constricted fibrous band be divided.

Preventive Measures

In the case of whites high shoes and finely woven stockings are the best protection. Native servants noticed with encysted chiggers should be compelled to remove them. All chiggers removed should be burned. Native children, who are generally affected, should be kept away from European quarters. As all domestic animals are attacked, such should not be sheltered near dwellings. Metamorphosis takes place in the dust and *débris* in cracks and corners, and sprinkling with kerosene or naphthol during the dry season will prevent chiggers from entering the house. In the case of ainhum, at the earliest onset of the disease, the native should be given shoes to wear and made to keep his feet entirely free from chiggers. I have seen incipient cases where this ended the disease. Grass cuts, etc., should be treated promptly and aseptically. Unaffected porters and servants who are expected to march through grass or in narrow paths should be provided with shoes or sandals.

6. CRAW-CRAW

This disease is a papulopustular skin affection and has been described from tropical Africa, India, and South China. Filariform embryos have been described as the probable cause of this affection. It is common all along the west coast of Africa, and all the cases seen by me were proven to be scabies. The severity ranges from cases in which a few slight papules may be found to almost incredibly severe and neglected cases, which may present all the symptoms enumerated in the descriptions of "craw-craw." In the worst cases large excoriations and ulcers are seen.

REMEDIAL MEASURES

Emily recommends thorough cleansing of the skin and its lesions, after which boric acid powder is dusted freely on the parts, which have been thoroughly scrubbed with sublimate lotion (1:1,000), and then boric acid vaselin is applied. Manson used warm carbolic acid lotion (1:20), followed by dry dressing with boric acid powder. Castellani and Chalmers favor the application of a 2 per cent. alcoholic solution of salicylic acid followed by a 5-10 per cent. β-naphthol ointment. Of course, where scabies is the factor, sulphur ointment is indicated.

7. SHIMA MUSHI

This disease is the Japanese river or flood fever. It seems to be confined to the banks of two rivers, the Shinanogawa and the Omoagawa, on the island of Nippon. The fever is supposed to be due to the bite of a larval mite resembling *Leptus autumnalis*. The nature of the germ introduced has not been determined, but a species of *Proteus* has been described as the causative organism.

The process is characterized by an initial eschar which afterward ulcerates, by fever, lymphangitis, and an exanthematous eruption. Bronchitis and conjunctivitis also occur. About fifteen per cent. of the attacks end fatally.

REMEDIAL MEASURES

The treatment is entirely symptomatic. Cold and antipyretics may be used to control the fever. The bowels should be kept open and the cough controlled by sedatives. Scheube points out that the Japanese react to antipyretics more strongly than Europeans, and recommends quinin and sodium salicylate in this disease.

PREVENTIVE MEASURES

Personal cleanliness and care of the skin, especially the genitals and armpits, are, of course, necessary. As the disease seems to affect the hemp-growing district of western Japan, it is easy for tourists to avoid this region, and consequently escape the bites of the Kedani mite.

8. ATRIPLICISM

This condition is stated to be due to poisoning by a plant belonging to the Chenopodiaceæ, and known as *Atriplex littoralis* (*A. serrata*). Other species of the same genus of plants are also stated to be poisonous. Some authors believe that a small insect found on the plant may be the cause of the effect seen in persons handling *Atriplex.* The disease is reported principally from China. With this affection may be mentioned the lacquer poisoning, which also occurs in China and Japan. Other inflammations of the skin are set up by the juice of different tropical species of *Rhus.* The species concerned in lacquer poisoning is *Rhus vernicifera.* Several poisonous species are natives of America.

Atriplicism is characterized by itching, edema, pain, local anesthesias, sensitiveness to heat, blisters, and sometimes ulcers. The symptoms of lacquer poisoning are similar to those set up by the juice of *Rhus toxicodendron,* familiar by personal experience to so many in the United States.

Remedial Measures

Local applications of cold, opium, and of chloroform are recommended in atriplicism. Lead and opium wash, lime water, or a solution of carbolic acid are advised in case of lacquer poisoning. Purgation in the beginning of the disease has been recommended. In weak individuals the dermatitis may be a serious matter, and good nourishment, tonics, and other hygienic measures are indicated.

9. LATHYRISM

This affection has been described from India, North Africa, and the Mediterranean districts of Europe. It is an intoxication, essentially a spastic spinal paralysis, and is due to eating *Lathyrus sativus* and other related Leguminosæ. The disease can be produced experimentally in animals. The use of this food for a period of one to several months is necessary to produce paralysis. The lateral columns of the spinal cord seem to be principally affected.

Remedial Measures

The food causing the mischief should, of course, be at once discontinued. Counterirritation over the spine, thermocautery, application of tincture of iodin, etc., are also recommended. Nourishing food and tonics and adequate clothing are indicated, and bromids have been advocated by several observers.

10. OTHER PLANT AND ANIMAL POISONS

Many poisonous substances are used by semi-civilized and savage races for purposes of arrow poisons, legal tests, murder, suicide, etc. I have described or reported some dozens of such substances from tropical Africa, and a large number have been studied in various other parts of the tropics. Some of the most important of these, with their antidotes, are here discussed.

A. Strychnos

One of the best known and most commonly used substances is Curare (also Warara, Urari, Wurrali, Woorari, etc.), active principle curarin. This is the South American Indian arrow poison obtained from different species of *Strychnos* (*S. toxifera, S. crevauxi, A. castelniœana, S. gubleri,* etc.).

Another substance with very similar action is Aker lampong, and also Ipoh Aker, the former from *Str. maingayi,* and the latter from *Str. wallichiana.*

The introduction of these poisons into the circulation by arrow wounds or otherwise is followed by swelling and pain at the point of puncture, elevation of temperature, increase in the amount of urine and the presence of sugar, slowing of the heart, gradual diminishing of respiration, and complete paralysis of the voluntary muscles.

Remedial Measures

Barbed arrows can be removed from the tissues by inserting grooved directors to cover the barbs. Ligate above the wound, which should be incised and sucked.

A solution of potassium permanganate should be injected around the wound, which should be washed out with the same solution.

Tannic acid rubbed into the wound is also recommended. Spirits of nitrous ether, one to two teaspoonsful in water, may be given. The victim should be catheterized to prevent reabsorption from the bladder. Heart stimulation with whiskey or brandy is advised by many observers. Some use a hypodermic of strychnin and atropin. Artificial respiration must be carried on, if necessary, and the patient kept warm with water bottles, etc.

B. STROPHANTHUS

The juice of this plant is used as an arrow poison in Central Africa, Dahomey, the Soudan, etc., and is variously called Kombe, Ine, Onage, Ochimbinga, etc. Some of the species used are *Strophanthus hispidus* (French Soudan), *S. glabrus, S. lanosus* (Zambezi and southeastern Angola), etc. This last is a variety studied by me.

Another set of substances known as Wabajo, Ouabio, Wabayo, etc., is obtained from various species of *Akokanthera* (*A. schimperi, A. wouabaio, A. deflersi,* etc.), and resembles *Strophanthus* in its mode of action.

The active principle of *Strophanthus* is strophanthin, a glycosid with the formula of $C_{31}H_{48}O_{12}$ (Arnaud). The active principle of *Akokanthera* is wabain, also a glycosid, the action of which is even stronger than that of strophanthin. These are violent heart poisons and cause death by paralyzing the heart in systole. The usual symptoms are weak pulse, increased amount of urine, increased muscular rigidity, spasms, etc.

Remedial Measures

The wound should be treated as in curare arrow poisoning, permanganate solution or tannic acid being used.

Chloral as an antagonist to the poison should be administered. The heart, circulation, and respiration should be stimulated with brandy or whiskey (two teaspoonsful every ten or fifteen minutes), aromatic spirits of ammonia, or caffein. A saline cathartic may be given. Inhalations of chloroform or ether to relax the muscles are recommended. Some place ice to the spine. Bromids and amyl nitrite have both been recommended. Artificial respiration should be practiced when the patient is *in extremis.*

C. ERYTHROPHLÆUM

Much has been written by travelers from time to time regarding the very weird and extraordinary custom practiced by the savage tribes of tropical Africa for the purpose of settling disputes among themselves, and usually known as the poison test. This unfortunate custom has nearly depopulated whole districts and has been responsible for more deaths in Africa than have many important diseases. The principal poison ingredient employed in this test is, in most parts of Africa, obtained from trees belonging to the genus *Erythrophlœum* (*E. judiciale, E. guineense*).

Other plants are also used either instead of or in connection with *Erythrophlœum.* Some of the most important of these are *Strychnos icaja, Physostigma venenosum, Tanghinia venenifera, Adenium somalense, Men-*

abea venenata, etc. The poison test is variously known in Africa under the names of Ombulungu, Ombambu, Wanga, Mulianti, Buave or Muavi, Mbundu or Mboundou, Ikazga, etc.

The poison observed by me is obtained from *Erythrophlœum guineense*, with which is usually mixed the inner bark of a shrub known as *Securidaca longipedunculata*, and the fruit of *Tephrosia vogelii.* The bark of *E. guineense* in small doses produces vomiting and purging; if powdered and inhaled it causes sneezing. The aqueous extract, when injected into the skin of animals, sets up vomiting and is said to irritate and afterward paralyze the vagus nerve, causing the heart to slow and finally to stop. I administered a piece of the bark two inches square and one-half thick, ground up and mixed with soup, to a small dog. Its limbs were paralyzed in twenty-one minutes, and it died in an hour and two minutes.

The active principle is erythrophlein and has been carefully studied by Lewin. The active principle of *Strychnos icaja* is also an alkaloid, akazgin, and resembles strychnin and brucin in its action. The active principle of *Physostigma venenosum* is variously known as physostigmin, calabarin, eserin, etc., and its effects are opposite to those produced by strychnin.

Remedial Measures

Emetics should, of course, be given. The heart weakness should be combated by stimulants. Permanganate of potassium is particularly indicated as an antidote to organic poisons in general, including all those mentioned in the preceding paragraph. It should be given in three- or four-grain doses dissolved in plenty of water at intervals of thirty minutes for a period of two or three hours.

If the poisoning is from *Strychnos* the patient should be placed in a horizontal position, the spasms controlled by inhalations of chloroform or ether, and the other well known remedies and devices against strychnin poisoning used.

In case the poison is from *Physostigma venenosum* the usual procedure in cases of eserin poisoning should be employed. Atropin is the best physiological antidote. In cases of poisoning by any of these substances, if breathing becomes labored, artificial respiration should be resorted to.

Preventive Measures

Native guides, etc., will generally warn the traveler of the poisonous character of the various herbs and plants encountered. The custom of administering the poisoning test should be put down by administrators and not winked at, even though, as is now often the case in Portuguese West Africa, modifications (the most common of which is probably the omission of the most dangerous ingredient, *Erythrophlœum*) are adopted in order that it be not invariably fatal.

D. VARIOUS ADDITIONAL POISONS USED FOR MURDER, SUICIDE, THE PRODUCTION OF ABORTION, ETC.

A large variety of poisonous substances are used by natives of warm countries for the purposes just named. Among these may be named snake venom, poisonous insects, e. g., *Epicauta ruficeps,* used in the Dutch East Indies for purposes of murder, *Diamphidia simplex,* employed by the bushmen of Africa as arrow poison, earth rich in the spores of tetanus bacillus, and putrid meat rich in ptomains, all used for the same purpose.

A large number of poisonous plants are also employed, of which only a few prominent species can be mentioned here. *Paullinia pinnata* is used in Brazil, the alkaloid known as timboin, and in the Dutch East Indies *Milletia sericea.* In Polynesia *Barringtonia speciosa, Nerium odorum,* etc., are employed in the same manner. In Africa various species of *Hyoscyamus* and several Euphorbiaceæ are commonly taken as medicine. As abortifacients *Cajanus indicus, Plumbago zeylanica,* the common nutmeg, etc., are in use in several tropical countries.

The effects of *Milletia* are characterized by violent pain in the head, tenesmus, and collapse. *Nerium* contains two glycosids, neriodorin and neriodorein, both of which are strong heart poisons. The African *Hyoscyamus* acts as the ordinary *H. niger* and contains both hyoscyamin and scopolamin. The Euphorbiaceæ have irritating and caustic properties, and in the case of one species used in West Africa death from collapse occurs in about an hour. The drastic substances used as abortifacients are, as a rule, not fatal, although *Plumbago* (active principle plumbagin) is very severe in its effects. The effects of the nutmeg (*Myristica fragrans*) are depressant, with headache, amblyopia, collapse, etc.

Remedial Measures

It is usually impossible to determine the exact nature of the substance giving rise to symptoms. Emetics or the stomach pump, when the poison has been swallowed, and ligation, excision, sucking, and cauterization when the poison has been introduced by means of a wound, are always indicated.

The most generally useful antidote is permanganate of potassium, due to the fact already pointed out that all of the known poisons employed by savage people are organic in nature. The permanganate should be given in solution in doses of two to four grains immediately after the emetic or stomach pump has been used.

In case of wounds the solution should be injected with a hypodermic syringe around the seat of the puncture. Heart stimulants and antispasmodics are, of course, to be employed when indicated.

Preventive Measures

It is never safe to accept food from savages unless these are known to be friendly. In traveling through unfriendly districts constant lookout should be made to avoid attacks with poisonous spears, arrows, or skewers set in the ground.

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CHAPTER IV

DISEASES DUE TO PROTOZOA

1. AMEBIASIS

WILLIAM B. WHERRY

INTESTINAL AMEBIASIS

When the encysted forms of certain protozoa of the genus *Entameba* reach the intestinal tract of man they burst, and the freed amebulæ (trophozoits) multiply in the gut contents. If they are saprophytic by nature their food consists chiefly of intestinal bacteria, and their presence and further multiplication may result in no harm to their host. However, if, through previous experience, the ancestors of these encysted forms have acquired a greater or lesser degree of parasitism, the young amebulæ start in on a parasitic existence, and the results to the host will vary according to the degree of parasitism acquired. The conditions favorable to the growth of amebæ in the intestinal tract of man seem to exist only in the lower ileum and colon.

Species of Ameba Concerned.—There is much discussion, chiefly between protozoölogists on the one hand and pathologists on the other, as to the importance of the presence of amebæ in the human intestinal tract. It seems to the writer that, considering the state of our knowledge of this group of microorganisms, due weight should be given to the contentions of both sides. The early work on amebæ and amebiasis is thoroughly reviewed by Musgrave and Clegg (9) (1904). Protozoölogists rightly contend that the early descriptions of species of amebæ are of no value, and that specific differences must be based on the method of reproduction, or this in conjunction with a study of possible pathogenicity. This point of view originated in the work of Schaudinn (1903), who adopted the generic name *Entameba* (Casagrandi and Barbagallo, 1895) for the amebæ parasitic in man. He described two species: *histolytica*, which he found in the stools of cases of tropical dysentery and considered the cause of this disease, and *coli*, which he found in the stools of a large percentage of "normal" individuals and considered a harmless parasite. Both species sometimes occurred in the same diseased intestine. The specific distinction was based, briefly, on difference in size, character of endoplasm and ectoplasm, nuclear structure, and method of reproduction. He was able to produce dysentery in kittens by feeding them encysted *E. histolytica,* but got negative results with *E. coli.* On this basis he established a pathogenic and a non-pathogenic species. That this may have been premature may be gathered from what follows. In addition to the above species, ten more have been described from the human intestine.

Doflein (2) has concluded that these various species may represent: (a) the descendants of the cysts of purely saprophytic species which have passed through the intestinal canal unchanged; (b) facultative parasites; (c) strict parasites.

Fantham (3) (1911) in a critical review concludes that we can accept but three fairly well recognized species, *coli, histolytica,* and *tetragena.* He tabulates the following characteristics:

E. coli.—Diameter 12-25 μ, but variable; no distinct ectoplasm, except at beginning of pseudopodia formation; nucleus round, vesicular, subcentral, with a karyosome; nucleus visible in life; multiplication by binary fission, schizogony—8 merozoits; reproduction by total and endogenous encystment, 8 spores in cyst.

E. histolytica.—Diameter 25-30 μ; ectoplasm distinct; endoplasm ingests red blood corpuscles; burrowing pseudopodia; nucleus small, eccentric, poor in chromatin, and invisible in life; multiplication by binary fission and by budding; reproduction by small exogenous spores.

E. tetragena.—Diameter 20-30 μ; ectoplasm distinct when pseudopodia are formed; endoplasm ingests red blood corpuscles; nucleus round and visible in life; multiplication by binary fission; reproduction by total endogenous encystment—4 spores in cyst. Viereck, who first described (1907) the *tetragena,* thought it a variety of *E. coli.* Further research may show this to be the case.

The Pathogenicity of These Species.—While *E. coli* is generally reported as being non-pathogenic, ulcerative colitis may be produced in cats by feeding them the encysted forms of *E. histolytica* and *E. tetragena.*

However, the writer agrees with Fantham that there has been a tendency to give undue prominence to Schaudinn's researches, and would further add that there has been an apparently wilful neglect of the significance of the cultural and passage experiments of Musgrave and Clegg. These workers were the first to cultivate a large series of amebæ, from man, in pure culture with a single, known species of symbiotic bacterium. Their extensive feeding experiments with amebæ in symbiosis with non-pathogenic bacteria were the first to convince us of the active rôle played by these protozoa. They not only demonstrated a wide variation in the pathogenicity of various cultures of amebæ, but showed that non-pathogenic, saprophytic strains could be made to acquire pathogenicity. It remains to be seen whether their work is not of great significance from the standpoint of prophylaxis. To the writer's knowledge this work has not been carefully repeated by anyone else, and from it we must conclude that there are not only strictly parasitic amebæ but also amebæ which have acquired varying degrees of parasitism. It is certain that further carefully controlled experiments on the adaptability of these protozoa would be welcomed.

It has been argued that, since the cultural forms do not exhibit the nuclear changes and methods of reproduction exhibited by forms studied in the stools, there is no connection between them. The recent work of Fantham upsets all previous studies. He has shown that the addition of some of H. C. Ross's "auxetics," leucin, tyrosin, etc., markedly stimulates nuclear division and schizogony in otherwise atypical cultures of *E. coli.*

Mode of Entrance and Exit of the Parasites.—After a period of existence in the cecum and colon, during which the amebæ multiply by simple fission, they encyst and escape with the feces. As Fantham has shown, a small amount of skatol causes the trophozoits to encyst rapidly. It is in this more resistant encysted stage that they succeed in passing the acid contents of the stomach of a new host. Water and uncooked foods act as the chief vehicles.

Werner (21) was able to show that *Ameba limax* multiplied and encysted in the intestinal tract of the house fly, and that the cysts passed out with its feces in a few hours. He obtained negative results with flies fed on stools containing the trophozoits of *E. histolytica.*

It is presumable that the cysts of parasitic species can be carried by currents of air. Wells frequently found amebæ (*limax?*) on plates exposed to the air of a jail in India.

Distribution of the Species and the Disease.—Although most prevalent in the tropics and subtropics, indigenous cases of intestinal amebiasis have been reported from many sections of the temperate zones. Patterson (10) has collected a list of cases reported as having acquired their infection in North America. Apparently many of these did not come in contact with previous cases. Long (6) reported a large number which acquired the disease in the neighborhood of San Francisco.

E. coli is ubiquitous and has been described from mice, men, monkeys, donkeys, etc. *E. histolytica* has only been found in man, and, according to Hartmann, is a rare parasite, the vast majority of cases of dysentery being, in his experience, due to *E. tetragena* (variety of *E. coli?*). Craig (1), Stiles, and others have reported the occurrence of the *histolytica* in North America. According to Hartmann (4) and Werner, the *histolytica* and *tetragena* have been found in cases from Asia, Africa, South and Central America.

Resistance of Amebæ to Various Destructive Agencies.—Most of our information is derived from experiments on cultural forms. When heated to 60° C. for one hour, some encysted cultures are killed, others survive. Marchoux (7) could no longer transmit dysentery to cats after warming stools containing amebæ to 45° C. for 35 minutes; and states that Kartulis, who confirmed his experiments in 1903, found that exposure for 24 hours in an incubator at 37° C. deprived dysenteric stools of all pathogenic action. Freezing to 12° C. kills some encysted cultures, while others survive. They are less resistant to light, for even the encysted forms succumb to the action of sunlight in about an hour; the Finsen light and ultraviolet rays exert a still more powerful action. Encysted cultures resist desiccation for periods varying from several months to six or more years. As a rule, they are more resistant to alkalies than to acids, though Musgrave and Clegg found a considerable variation in the degree of resistance. Quinin hydrochlorate in 1:2,500 solution kills the trophozoits in 10 minutes. Quinin and silver nitrate killed amebæ in 1:20,000 and 1:300,000 dilutions respectively some time between one and three days. A slightly acid solution of benzoyl-acetyl-peroxid (acetozone) is fatal, in 1:5,000 solution, in 10 minutes.

Vedder (17) has reported that certain preparations of ipecac exert a marked lethal action on cultures of saprophytic amebæ, paramecia, and balantidia. The efficacy seems to depend upon the emetin content, and this fact points to the greater excellence of the Brazil root. While several preparations killed amebæ in dilutions of 1:10,000 to 1:50,000, a preparation of deëmetized ipecac failed to kill in 1:5,000 solution. Emetin alone killed the ameba, paramecium, and balantidium in 1:100,000 solution. He suggests that one should insist that the ipecac used contains the required amount of emetin, or insist upon obtaining the Brazil root. Vedder also shows that two per cent. of the fluid extract of ipecac exerts a marked inhibitive and germicidal action on *B. typhosus, B. paratyphosus, B. dysenteriæ,* and the *Staphylococcus pyogenes aureus.* This is of importance when one considers the likely rôle played by symbiotic bacteria in amebic lesions.

The writer has repeated part of Vedder's experiments. The action of Merk's emetin was tested on a saprophytic ameba, grown from tap water in symbiosis with a single species of non-spore-bearing bacillus. Especial control experiments showed that, under the conditions of the experiments, the lack of symbiotic bacteria and the action of body temperature did not influence the results. Emetin, even in 1:200,000 dilution, killed the trophozoits in 24 hours at 36° to 38° C. A single hour's exposure was insufficient even in 1:20,000 dilution. The encysted forms were much more resistant, but, as amebæ do not encyst in the tissues, this factor may be disregarded.

The Rôle of Symbiotic Bacteria.—There seems to be a tendency to overlook the probably important action of the bacteria which usually accompany the amebæ into the deeper layers of the intestine and thence to the liver, brain, and elsewhere, in cases of generalized amebiasis. The fact that when amebæ occur at these sites without bacteria they cannot be cultivated, whereas they can when bacteria are present, is significant. As Musgrave and Clegg maintain, the change from a tissue symbiosis back to a bacterial symbiosis may be a much more difficult step than the reverse process. Protozoölogists have largely ignored this phase of the subject, and it still remains to be shown that the initial lesion, superficial necrosis of the intestinal mucosa, is not due to the action of amebæ and bacteria. The species of bacteria which accompany amebæ in abscess of the liver are *staphylococci,* undetermined anaerobes, bacilli of the *coli* group, and *streptococci* (Rogers, Marchoux, 1908). Rogers has attempted to show that the presence of bacteria in liver abscesses is due to post-operative infection, but fails in his reasoning to take into account that the changes produced by drainage will favor the multiplication of any facultative anaerobes already present and so enhance the chances of their subsequent recovery in cultures. This, however, if true, does not negate the value of his method of treating abscess of the liver. Marchoux by means of serial passages succeeded in infecting 82 cats. Fifty-two of these died with abscesses of the liver, and all of these abscesses contained *staphylococci,* various anaerobes, bacilli of the *coli* group, *streptococci,* and *Proteus vulgaris* in association with the amebæ. Musgrave and Clegg report similar results in experimental amebiasis in monkeys, but the species of bacteria concerned are not mentioned.

General Nature of the Disease.—The parasites in the vast majority of cases gain entrance through the mucosa of the cecum or colon, where acute but more commonly subacute or chronic ulcers are produced. In a careful analysis of 200 cases, studied post mortem, Woolley and Musgrave (22) found the entire large bowel involved in 159; the cecum and ascending colon alone in 23. In 7 of the total cases the ileum was involved just above the valve; and in 14 the appendix was ulcerated. The lesions may vary from small raised hemorrhagic spots, which later lose their epithelial coverings and ulcerate, to sharply circumscribed or, more commonly, undermined ulcers which may vary greatly in size—from a few millimeters to several centimeters in diameter. Ulceration may be accompanied by hemorrhage, diphtheritis, or gangrene, and perforation may result in local or general peritonitis or retroperitoneal abscesses which may burrow widely. The connective tissue of the submucosa offers the least resistance to the process—hence the undermining of the mucosa. Secondary bacterial infections undoubtedly aid in the progress of the ulceration and may be an important factor in lowering the great resistance of the mucosa and muscularis.

The amebæ, accompanied by or without symbiotic bacteria, may reach the liver through the portal circulation, and there produce single or multiple abscesses. Musgrave has twice found amebæ in the peripheral circulation, once associated with and again without bacteria. Ulceration may or may not manifest itself by dysentery. While diarrhea, with or without the presence of mucus or blood, is a prominent symptom which appears sooner or later in the course of the disease in a large percentage of the cases, one should bear in mind the fact that severe infection may exist without the production of any marked symptoms. Such cases have been reported by Osler, Dock, Councilman, Laffleur, Kartulis, Tuttle, Rogers, and others, and Musgrave has reported fifty fatal cases. Similar cases probably have been included in the statistical studies of healthy individuals with amebæ in their stools as reported by Craig and others.

This brings up the much disputed question as to whether the mere presence of amebæ in the stools is sufficient evidence of infection to justify treatment. The work of Wenyon (20) on the *E. coli* of mice shows very clearly that dietetic disturbances resulting in diarrhea may wash out such large numbers of amebæ, which here are apparently harmless commensals, as to make one think that they were the cause of the diarrhea. It can hardly be doubted that this may also occur in humans, and yet it seems hardly fair to the host to leave the decision as to the pathogenicity of the amebæ to the physician when one considers the state of our knowledge of the species and their adaptability. It is far safer, as Musgrave has so often reiterated, to apply the therapeutic test: If the amebæ are harmless commensals, they can be removed with comparative ease; if harmfully parasitic, they may or may not be dislodged after prolonged and careful treatment.

Treatment

Hygienic Treatment.—Any routine treatment indiscriminately applied will often fail. The general condition of a case, its duration, the site of the lesions, the presence or absence of complications, may call for varying remedial measures. As a general rule, if acute dysenteric symptoms are present, the patient should be put to bed, kept on a fluid diet of milk and eggs, the pain and dysentery checked with opium or morphin.

Cases without acute symptoms, or when these have ceased, rarely require rest in bed and usually do better when on the treatment by rectal injections, on a moderate amount of exercise, and a full diet. The food, however, should not be of such a character as to leave much residue. When patients are emaciated or anemic they should be systematically weighed and their blood should be examined at intervals.

Drug Treatment by Mouth.—Many English practitioners have long held that ipecacuanha is as great a "specific" in dysentery as quinin is in malaria. The types of dysentery were not clearly differentiated in the earlier days, and it remained for Rogers to point out the beneficial effect of ipecac in the presuppurative stage of amebic hepatitis. The work of Vedder, already cited, on the amebacidal action of emetin would seem to support this clinical and post-mortem evidence. Dock, Futcher, Simon, Long, and others in this country report on rapid improvement following the ipecac treatment. It is recommended that the ipecac be given in doses varying from 20 grains (1.3 gms.) morning and evening to 5-10 grains (0.325-0.65 gm.) three times a day, in salol-coated capsules. The coat upon the capsules should not be thicker than one-eighth of an inch. If the capsules pass through undissolved the thickness of the coating may be reduced to one-sixteenth of an inch. The patient should be at rest in bed and kept on a milk diet throughout the period during which ipecac is given, and the drug is best borne after a fast of about three hours. These large doses are given daily for a week or more, or until amebæ can no longer be found in the stools, when the dose is reduced to 5-10 grains (0.325-0.65 gm.) daily throughout convalescence. Only repeated examinations of fecal specimens, obtained after a dose of salts, should convince one of the absence of amebæ. It is well to examine the stools of a patient for a month or more after apparent recovery.

The favorable reports on the use of bismuth, salol, etc., are unsatisfactory, as the apparent cures wrought were not sufficiently controlled. It may be stated, however, that bismuth should not be given during local treatment by means of enemas.

Local Treatment by Enemas.—Much relief can be given and cure often accomplished by systematic irrigation of the large bowel with solutions of quinin 1:1,000 to 1:500; potassium permanganate 1:1,000; hydrogen dioxid, 25 per cent. solution.

As a rule, it is best to delay local treatment until the acute symptoms have been allayed. On the other hand, when the bowel is extremely irritable, it may be prepared by an enema of two liters of warm water, containing ¼-½ grain (0.0167-0.0325 gm.) of morphin, administered one-half hour before the quinin, etc. The strength of the amebacidal agent used in solution will vary with different cases. Quinin 1:1,000 is well borne by subacute cases, and this may be gradually increased to 1:500. The temperature at which a quinin solution is best borne and retained varies with different cases; some prefer it cold, others delivered at body temperature. The solution is best administered from a glass irrigator of at least two liters capacity through a soft rubber connecting tube, which in turn is connected by a valve stopcock with a red rubber rectal tube at least 100 centimeters in length and from 10-15 millimeters in diameter. The latter must be of moderate firmness with the opening at the end of the tube.

With the patient in the knee-chest position, or in the Sim's position with the hips well elevated, the well-vaselined-filled tube is inserted into the rectum. After a few moments have elapsed to allow the spasm of the sphincter to subside, the fluid is turned on slowly and the tube gradually inserted along the path made for it by the fluid. As much fluid as can be borne should be run in and the patient encouraged to hold it as many minutes as possible. After the fluid has been administered, often a change of position will enable the patient to hold it longer. It is important that the fluid fill the cecum. The frequency with which this treatment may be given varies, but, as a rule, once daily is enough because of its severity. In the average case it is well to stop the rectal injections in two weeks, and after a day's rest to administer a dose of salts. The stool so collected is examined for amebæ. If they are still present treatment is resumed and kept up, until after repeated examinations one is assured of the disappearance of the parasites. No doubt many cases yield to careful, persistent, controlled treatment along these lines. However, to stop treatment upon the cessation of symptoms is foolish. Only repeated examinations covering weeks or months after the cessation of treatment should convince one that the parasites have disappeared. According to the severity of the process, such treatment may have to be repeated for weeks or months. Often patients who have not been cured by such treatment in the tropics will improve and recover under the same treatment upon return to a temperate zone.

Vincent (18) recommends colonic flushing with one-half per cent Labarraque's solution (sodium hypochlorite). He says that experimentally this solution killed the *"Entameba"*; that the stools of 5 cases after treatment became normal in 24 hours; in the rest of 18 cases the amebæ disappeared in 2-4 days.

HEPATIC AMEBIASIS

Prophylaxis

It is not entirely clear why hepatic lesions do not occur more frequently when amebæ can be found often, post mortem, in the portal blood vessels. Undoubtedly, systematic treatment of the intestinal lesions instituted early in the disease tends to prevent amebic hepatitis, as illustrated by the almost complete disappearance of this complication in the Philippines among cases systematically treated by enemas.

In 1907 Rogers pointed out that the formation of liver abscess could be detected in the presuppurative stage by the presence of a marked leukocytosis with little or no increase in the proportion of polynuclears. When suppuration in the liver has taken place the proportion of polynuclears rises, sometimes to 90 per cent. of the leukocytes present (Rogers, Léger). He believes that in this stage an abscess may be prevented from forming by giving ipecac in 10-grain (0.65-gm.) doses three times a day. The writer has seen hepatic tenderness and leukocytosis accompanying intestinal amebiasis disappear rapidly in cases so treated by J. D. Long in the Marine Hospital at San Francisco.

Treatment

The type of abscess present affects the prognosis, as shown by the following table compiled from post-mortem records by Rogers (1908):

Classification of Fatal Operation Cases of Amebic Abscess

	Cases	Percentage	
Cases with serious complications such as secondary, large abscess of the lungs	2	5.7	51.4*
Very acute with no limiting wall and multiple abscesses	10	28.6	
Very large single abscess in exhausted subjects	6	17.1	48.6†
Single small and medium sized abscesses limited by a fibrous wall	11	31.5	48.6‡
Single fibrous walled abscesses with very few minute secondary ones	6	17.1	

* Very fatal. † Single abscess. ‡ Recoverable.

Since the most hopeless cases are those with non-circumscribed, rapidly spreading, multiple abscesses, it will be seen that about one-half of these should have recovered under appropriate treatment, barring serious secondary infection. The *Streptococcus pyogenes* is the most virulent secondary invader.

On the basis that post-operative sepsis carries off many otherwise hopeful cases, Rogers suggested the abolition of the open method of operating and the adoption of a combined aspiration and irrigation needle devised by himself. By means of this the abscess cavity is irrigated daily with sterile bihydrochlorate of quinin solution, 5 grains (0.325 gm.) to the ounce (32 c. c.), in order to destroy the amebæ present in the wall of the abscess. He reports a very successful case so treated by Murray (Rogers, 1908), and has further summarized his results to date (Rogers, 1910). Microscopic and bacteriologic examinations of the aspirated pus will furnish data concerning prognosis and the progress of the case.

Exploratory Puncture in the Diagnosis of Liver Abscess.—This is not without danger, for fatal hemorrhage sometimes follows the operation. The blood count will indicate whether suppuration has occurred or not. In any event, unless the abscess can be easily detected on physical examination, the patient should be put on full doses of ipecac (twenty grains—1.3 gms.—morning and evening, or five grains—0.325 gm.—three times daily) for a week. This treatment will delay further suppuration, and by reducing hyperemia lessen the danger of exploratory puncture.

Apparently the use of chloroform is particularly dangerous in operations for liver abscess (see Hunter).

After-treatment.—Rogers urges that every patient operated on for liver abscess be put on full doses of ipecac as soon as possible to heal the intestinal lesions, which are often latent or so situated as to produce no symptoms.

2. MALARIA

Creighton Wellman

The term malaria is here used to indicate a large and important group of conditions depending upon the presence in the blood and tissues of certain sporozoan parasites, and characterized by periodicity in the symptoms, deposit of peculiar pigment in the parenchymatous organs, especially the spleen, amenability to quinin, and dependence for spread upon certain groups of mosquitoes.

The great importance of malarial infections in the tropics renders it desirable that the principles and details of effective treatment be made clear.

The malarial parasites are sporozoan protozoa of parasitic habits which live in the blood and tissues of vertebrates during schizogony, and in blood-sucking arthropods (mosquitoes) during sporogony. In the course of this cycle the vertebrates are intermediate hosts and the arthropods the definite hosts of the parasite.

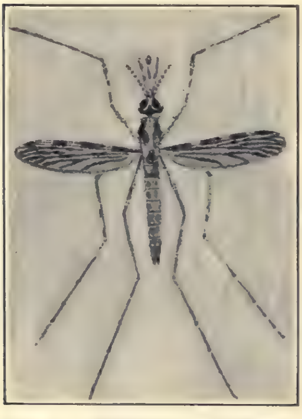

Fig. 1.—Myzomyia Funesta, the Principal Malaria-bearing Mosquito of Tropical Africa. (After Wellman.)

The species infesting human beings all belong to the family Plasmodidæ. In this group the schizont lives in or on the erythrocytes and forms rosettes. The sporont lives in Culicidæ, where it undergoes a coccidia-like cycle in the stomach of the mosquito, resulting in many sporozoits, which are freed and reach the salivary glands of the insect and thus enter the intermediate host. Three species belonging to the genus *Plasmodium* are usually described as representing the parasites of human malarial disease. The diagnosis of these is briefly as follows:

(1) *Plasmodium vivax.*—The grown schizont averages about 10 μ in diameter, has its nucleus at the edge, the hemozoin is in fine particles, the schizogony occurs in the peripheral circulation, the cycle occupying about forty-eight hours. The merozoits are from fourteen to twenty or more, and the young schizonts are very active. The host cells are greatly swollen and pale, and when stained with Romanowsky show the basophilic stippling known as Schüffner's dots. The gametocytes are round, aver-

age about 14 μ in diameter, and their hemozoin is in coarse granules. The sporogony takes place in Anophelinæ. This is the benign tertian parasite. There are many species which occur in other vertebrates that are similar to this and the following ones.

(2) *Plasmodium malariæ.*—The grown schizont is smaller than a red blood corpuscle, and the hemozoin is coarser than in *P. vivax.* The schizogony also takes place in the peripheral circulation, the cycle occupying about seventy-two hours. The merozoites range from six to twelve or more, and the young schizonts are smaller than those of *P. vivax.* The host's cell is smaller and darker than a normal stained corpuscle. The gametocytes are round, seen rather rarely, and are irregular in size, sometimes being smaller than red blood corpuscles. The sporogony is in Anophelinæ. This is the quartan parasite.

(3) *Plasmodium falciparum.*—The grown schizont is less than half the size of the erythrocyte. The hemozoin is very fine. Schizogony occurs in the internal organs, the cycle occupying about forty-eight hours. The merozoites number from fourteen to thirty, being very irregular in number. The host cell, as seen in stained specimens, is sometimes shrunken and changed in color, being a green or brassy hue. The gametes and gametocytes are crescentic in shape, and average about 10 μ by 2.5 μ. Sporogony is in Anophelinæ. This is the estivoautumnal or malignant parasite.

A fourth species, supposed to be the cause of quotidian or estivoautumnal malaria, is described by various writers. This species is said by some authors to be unpigmented. In West Africa I have never been able to demonstrate such a form, although Craig and others, working in the far East, are convinced that the quotidian species should be recognized. Of course, double infections often occur.

The conditions set up by the presence of the parasites are myriad. The various types and cases will later be mentioned more in detail, and the masked forms dealt with. As a general rule, among the prodromal symptoms pain in the shins, yawning, and headache are those of which complaint is most frequently made. Rigor is generally slight, often absent. A sense of suffocation is common. Epistaxis and vomiting are occasionally seen. The temperature sometimes runs high and, in tropical African estivoautumnal infection, is very often of a remittent type. The spleen is almost always inflamed, or at least congested, even in comparatively mild attacks. In common with other observers, I have often demonstrated albuminuria.

Many of the apparently healthy natives of malarial regions have a high percentage of large mononuclear leukocytes, and I have sometimes noticed the presence of myelocytes during acute attacks. I generally have found the leukocytes absolutely diminished, but not always so, as is claimed by some observers, and the number of erythrocytes and hemoglobin index are often higher than one would judge from the appearance of his patients. The tongue is often foul, the bowels constipated, and the appetite poor.

I have recognized the following clinical types in my studies in West African estivoautumnal fevers: (1) Comatose. This type is characterized by restlessness, high temperature, pains in the head, delirium, final coma, and not seldom death, especially when the physician is not called early in the attack. (2) Hyperpyrexial. The principal features of this form are very high temperatures. In tropical Africa care must be used to avoid confusing this type with so-called "hyperpyrexial fever," which may be non-malarial and in which I found on one occasion a bacillus in the blood. (3) Syncopal. In this form the patient collapses and death is sudden from heart failure. (4) Typhoid. This type is not uncommon in the tropics, especially in West Africa. From a typical malarial attack, which in the beginning may be very acute, the patient runs into a typhoid condition in which he may remain for a long period. This form is differentiated from enteric fever by its amenability to full doses of quinin. Mixed infections must be carefully diagnosed and treated. (5) Mild or walking cases. These are not uncommon and occasionally do not go to bed or apply for treatment. Fortunately, in the tropics we see a good percentage of such cases. (6) Gastric. These types are often like the walking cases except for bilious vomiting and gastric distress. The temperature may be only very slightly raised.

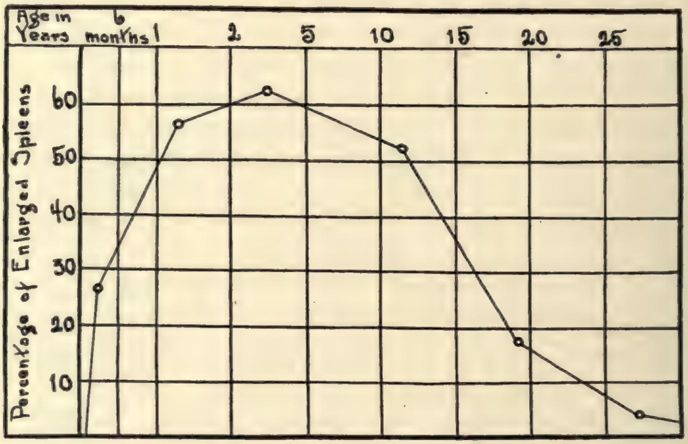

FIG. 2.—CHART SHOWING SPLEEN INDEX OF 513 UNTREATED NATIVES OF WEST AFRICA (351 children and 162 adults). (After Wellman.)

There have been described from the tropics by various observers other clinical types of estivoautumnal fevers, but it seems hardly necessary to describe these here, as they are usually of the pernicious variety, and the malarial parasites are, as a rule, abundant in the blood, rendering their diagnosis easy. Among such types may be mentioned the delirious, hemiplegic, algid, hemorrhagic, and pneumonic forms recognized by systematic writers.

Atypical malarial fevers are numerous in the tropics, and masked forms, or the mimicking of other diseases, are not uncommon. Some of the most frequent of these forms seen in West Africa are: (1) Dysenteric. This variety cannot be told from acute dysentery clinically. The temperature may be slight. (2) Low even temperature simulating that from septic processes. This form might be confused with tuberculosis, liver abscess, etc. (3) Epileptiform convulsions. These are especially common in children, and are very like the fits caused by ascariasis. (4) Malarial ulcers. These are round, punched-out, discrete ulcers with slightly raised edges.

Various writers describe many other atypical and masked forms which are liable to be confused clinically with appendicitis,[1] insanity, muscular rheumatism, cerebrospinal meningitis, bronchitis, neuralgia, etc.

Some of the most frequent complications in my experience are abscess of the spleen, abortion and miscarriage, amblyopia, orchitis and hydrocele, intermittent colic, and aphasic, multiple peripheral neuritis, pruritis, and numerous other nervous manifestations.

It must never be forgotten by those practicing in tropical countries that almost any infectious disease may stir up a latent malarial infection and that then we have two coincident processes to recognize and deal with. In such instances the malarial process may be entirely masked by the other disease. I have found amebic dysentery, liver abscess, pulmonary tuberculosis, acute bronchitis, acute articular rheumatism, typhoid fever, appendicitis, and various other diseases to play such a rôle in cases of Europeans in tropical Africa.

While the manifestations of estivoautumnal malaria are so protean and really defy classification, the tertian and quartan fevers show more conformity to type and the clinical picture is, as a rule, well marked and unmistakable.

The paroxysms of tertian malaria, if untreated, take place with cyclical regularity every forty-eight hours during the course of the disease. There is usually a chill, followed by a hot stage, which, as a rule, terminates in a remission or stage of sweating.

In quartan infections the attacks, of course, occur at intervals of about seventy-two hours. It need hardly be pointed out again that double, or, more rarely, multiple, infections may obscure the picture, and irregular, quotidian, or even remittent fevers may be seen. In some instances the sporulation of the parasite may cause few or no symptoms.

The condition of the patient and his symptoms depend upon the species and development stage of the parasite with which he is infected. In tropical Africa I found only fourteen cases of quartan infection in 531 blood examinations for malaria. Benign tertian was not seen at all, although this last form is not uncommon in other parts of the tropics, notably in the Philippines. Thus we see that in tropical Africa, and the same is true of many other parts of the tropics, the estivoautumnal form

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[1] In one case that was recently referred to the writer the patient was on the table prepared for laparotomy, when a thick film blood preparation showed the presence of malaria. A few hours after an intravenous injection of quinine the patient was sitting up in bed eating his breakfast and later made an uninterrupted recovery.

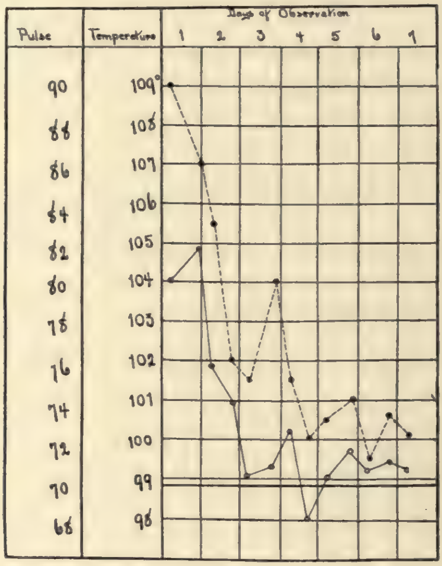

FIG. 3.—COMBINED PULSE AND TEMPERATURE CHART OF AN UNTREATED CASE OF QUARTAN MALARIAL FEVER ENDING IN SPONTANEOUS RECOVERY, OBSERVED IN A WEST AFRICAN NATIVE. (After Wellman.) The continuous line indicates the temperature in the mouth, the dotted line represents the pulse.

is by far the most important, and has, indeed, some claim to be known as the "tropical parasite."

While the technique of the laboratory diagnosis lies without the scope of the present discussion, yet it cannot be too strongly insisted upon that careful microscopical examination of the blood should determine the character of the infection and the time when the parasite can best be attacked by therapeutic measures.

The pathology of the disease consists principally of an anemia due to diminished volume of the blood elements, and of hemoglobin value. The oligocythemia is marked. The leukocytes are, as a rule, absolutely decreased, but there is usually a large mononuclear leukocytosis. The spleen and liver are usually enlarged and of a dark slate color, the spleen especially, and in chronic cases amyloid degeneration may be seen. The brain and bone may be pigmented, the former with punctiform hemorrhages.

REMEDIAL MEASURES

The history of malaria therapy could well be divided into two great periods: (1) from the most ancient times to the discovery of cinchona bark, and (2) from the discovery of cinchona bark to the present time. For, as will be brought out later, the treatment of malaria spells quinin.

The objects to be gained by treatment are the eradication of the causal organism from the patient and the relief of the patient from the injuries from which he has suffered as the parasite's host. In tropical countries this can rarely be completely accomplished. The most that can be hoped for is continued latency. The manner by which latency is obtained is not understood. The natural resistance of the patient no doubt plays a large part in the matter. The persistence of the parasites, even after long-continued treatment, has been a matter for much conjecture and study. Craig's theory of the intracorpuscular conjugation of the parasites has not been accepted by many workers. For instance, Cropper seems to have shown that, when double or treble infection occurs, instead of conjugating, each parasite goes on to full development. Could light be thrown on this difficult point, the therapeutic eradication of malaria from the body would present fewer difficulties.

Specific Treatment.—As has just been said, many drugs have been tried and recommended in the treatment of malaria, yet, in the words of Manson, "all sink into insignificance in comparison with quinin." This is truly specific in its action, in that it destroys the causative organism of the disease. But it must always be kept in mind that, especially in severe infections, the drug must be given promptly in sufficiently large quantities, and the physician must satisfy himself that the drug is absorbed. Another point that should never be forgotten is that quinin does not exert its full action if the liver be engorged, and consequently calomel, or some other efficient so-called hepatic stimulant, should be given with or preceding the administration of quinin.

Solubility, absorbability, and the power of being eliminated by the patient are all qualities which must be taken into consideration in quinin therapy. According to MacGilchrist, experiments on animals have shown that the absorbability of quinin by the several modes of administration is in the following order: (1) subcutaneous injection in extreme dilution (hypodermoclysis); (2) oral administration during fasting; (3) oral administration with, or immediately after, taking food; (4) subcutaneous injection in the strengths usually employed—1 in 2 up to 1 in 8 (ordinary hypodermatic or intramuscular injection). The most interesting point brought out by these experiments is that oral administration apparently affords more prompt action of the quinin than subcutaneous administration in low dilutions.

Of course the rapidity of the absorption depends upon the salt employed and the method of administration. Deaderick has compiled the classical literature on these points, and states that the drug begins to appear in the urine in the following lengths of time: When intravenously injected, in 10 minutes; when intramuscularly injected (variable according to dilution); when injected per rectum, in 20 to 25 minutes; when given by mouth, hydrochlorid, in 15 minutes; bisulphate, in 30 minutes; acetate, in 30 minutes; citrate, in 30 minutes; sulphate, in 45 minutes; tannate, in 3 hours.

While, as MacGilchrist states, quinin is more slowly absorbed from a full stomach than an empty stomach, yet it has been demonstrated by Mariani that during twenty hours after the administration of the drug the absorption is greater from a full stomach than from an empty one.

Another interesting observation is that of Giemsa and Schaumann, who found that the insoluble or slightly soluble salts, when given by the mouth, are as rapidly and completely absorbed as the more soluble ones.

Quinin is excreted in the urine, the feces, and in mothers' milk. It is also found in the amniotic fluid and in the urine of newborn children of mothers who are taking the drug.

Choice of Preparation.—In choosing a preparation I believe that the more soluble salt should be preferred, even when it is intended to administer it by the mouth. However, the exact object to be attained should weigh in the decision of this matter.

The most commonly used salts are the following: (1) *Sulphate.* This preparation is popular and cheap, but is, in my opinion, the least desirable of all those here mentioned. Most of the gastrointestinal irritation noticed in quinin users is attributable to the effects of this salt. (2) *Bisulphate.* This is a good and soluble preparation, much preferable to the sulphate. The same may be said for the hydrobromid and hydrochlorid. (3) *Bihydrochlorate.* This is a fine, soluble salt, and, in my experience, has given the best clinical results of any. (4) *Ethyl-carbonate* (euquinin). This form, as well as the tannate, while slightly soluble, is of value in treating children on account of its tastelessness. (5) *Acetyl-salicylate.* This new salt is mentioned here, as it may be of use in special malarial conditions, since its effect is only obtained after it reaches the alkaline contents of the intestine.

The exact solubility and per cent. contained of alkaloid are not, in my opinion, matters of great importance in choosing a preparation for administration. Here, as in many other circumstances, the clinical test is the crucial one.

The following table exhibits the solubility and alkaloidal value of the most important salts of quinin:

	Solubility in Parts of Water	Per Cent. of Alkaloid
Acetate	Slightly	84
Acetyl-salicylate	?	?
Anhydrous quinin	1.750	100
Arsenate	Slightly	69.4
Bichlorid of quinin and urea*	?	?
Bihydrobromid*	7	60
Bihydrochlorid*	1	72
Bisulphate	8.5	59
Citrate	820	67
Ethyl carbonate (euquinin)	12,500	81
Hydrobromid*	40	76
Hydrochlorid	18	81
Hydrochloro-sulphate*	2	74.3
Lactate*	10	78
Nucleinate	Insoluble	?
Phosphate	420	76.2
Salicylate	77	68
Sulphate	720	74
Tannate	800	30
Valerianate	53	76

The salts that are best suited for subcutaneous administration are marked in the table by an asterisk.

Time of Administration and Action on Cause of Disease.—In regard to the time of administration, it may be said at once that in severe cases the physician should not wait for any particular phase in the sporulation of the parasite. For, in the treatment of children especially, and of severe attacks in Europeans, it is highly important that quinin should be administered *promptly* in order to save life.

It is hardly possible to eradicate the disease on the occasion of the first treatment, however thorough that treatment may be. Consequently the procedure should be repeated in thirty to forty days from the date of an attack. This should not only be done in malarial countries, but also where the patient has returned to a healthy climate, as liability to recrudescences must be kept in mind, as well as the possibility of reinfection.

As regards the relation between the time when quinin is administered and the influence of the drug on the symptoms of the disease and the condition of the parasites, the original observations of Marchiafava and Bignami have never been surpassed. These authors found the following to be the facts: (1) If the drug is administered within the six hours preceding the attack, it has no immediate influence on the course of the disease (or, at most, only slightly delays and lessens the attack), the parasites tending to form gametes; the subsequent attack usually does not occur. (2) If the quinin is given at the onset of the attack, the attack will usually come on; a subsequent attack is usually prevented. (3) If the drug is administered during the febrile stage, the attack is not appreciably modified, although the subsequent attacks may be prevented or rendered very mild. (4) If the quinin is given near the time of crisis, the parasites still persist in the blood. (5) If the quinin is administered at or after the crisis and continued during the apyrexia, the next attack is prevented or rendered abortive.

From these and a large number of more recent investigations, it is plain that the stage of the parasite most vulnerable to the action of quinin is the merozoite, before it has attacked the red blood corpuscle. Therefore, it is essential to produce in the blood serum as strong a solution of quinin as possible against the time of sporulation, "that the young parasites may be born into a toxic medium" (Deaderick). It may be added that clinical experience is in entire accord with this doctrine. It must not be forgotten, however, that the sporulation of the parasite is more or less irregular and continuous, and consequently divided doses given at regular intervals are also useful in stopping this promiscuous reproduction.

To sum up: Torti's method of giving the quinin before the paroxysm is inferior to the method of Sydenham, which is to give the drug in the decline of the paroxysm. With this latter method there should be combined the administration of small fractional doses in order to destroy the irregularly sporulating parasites between attacks.

The action of the quinin on the malarial parasites has been studied by many authors from Laveran to the present day. The observations of Craig are admirable in this connection. The quinin-poisoned young ring forms may be recognized by their taking stains very intensely. Later fragmentation of the parasite is seen to begin. Still later, after the parasites are from one-half to three-fourths grown and pigment formation has begun, the fragmentation produced by the quinin is much more marked. In such parasites a non-staining area may be observed around the chromatin, and even extrusion of the chromatin from the plasmodia may be demonstrated. Extracellular parasites are seen, as well as instances of incomplete segmentation and other evidences of degeneration. In the case of estivoautumnal parasites only the young ring forms are, as a rule, seen in the peripheral circulation, but these after quinin are distorted and broken down.

The gametes often seem to be not only uninjured, but even encouraged, by quinin, especially in small doses. In fact, small doses of the drug are, in the writer's experience, able to arouse latent malaria and to precipitate an attack. At any rate, such amounts only serve to defeat our purpose by forcing the parasite from schizogony into a sporogony that is not amenable even to the largest doses. On the other hand, adequate amounts of quinin tend to reduce the number of crescents or other gametes, probably by killing the younger forms from which such gametes are thought to be derived.

Dose and Mode of Administration.—As has just been said, it may be pointed out, as a cardinal principle in quinin administration, that it is essential to give a large dose *at first* so as to destroy the parasites. As has been said, small doses in the beginning of treatment only serve to force the parasite into sporogony, in which state it becomes inaccessible to the drug; for quinin kills the schizonts, but does not readily destroy gametes. These latter will, however, disappear from the blood in time if reinfection is prevented.

Various modes of administration have been practiced, e. g., by the skin, mouth, rectum, and hypodermically. This latter may be subcutaneously, intramuscularly, intravenously, intraviscerally, intraspinally, etc.

In tiny infants I have usually given it *per rectum*. Generous doses must be employed with this method. I seldom give small children quinin by the mouth.

Taking up these methods severally, it may be said that oral administration is by far the most usual method, and, unless the patient is in a critical state, is a very effective one. An important point, especially in the tropics, is that quinin should always be administered in solution or in powder. Capsules and wafers may be allowed, but pills and tablets of quinin are, in my experience, frequently passed unchanged by the patient.

As regards the dose, using the bihydrochlorate as a standard, it seems to be clear that a dose of 20 to 30 grains (1.5 to 2.0 gm.) with later doses of from 10 to 20 grains (0.6 to 1.3 gm.) every four hours will clear up an ordinary attack in from one to four days. Rogers recommends only 10-grain (0.6-gm.) doses three times a day, and considers that larger amounts are not any more efficacious.

My own opinion is that the larger doses are necessary, especially in estivoautumnal infections in tropical countries, and, unless the patient's digestive functions are active, even these amounts are not effective when given by the mouth.

Coming now to the fractional doses to be given between attacks, it may be said generally the alkaloid may be administered at the rate of one grain (0.065 gm.) per hour in the case of an adult weighing one hundred and fifty pounds. This amount may be given either in 2- or 4-grain (0.12-gm. to 0.25-gm.) doses 2 or 4 hours apart, as seems best. [In the temperate zones we find all forms of malaria, but the majority of cases are caused by the tertian and quartan parasites, the minority by the estivoautumnal type. As a rule, the milder forms prevail, and it follows logically that the ordinary dosage and the ordinary mode of administration will suffice to overcome the disease. In intermittent fever I give from gr. xv-xx, children in proportion, to be taken within a half hour, and usually between six and half-past six in the morning, this to be repeated every seventh day for four weeks from the day the last paroxysm has occurred. In other respects malaria of the temperate zones is treated like Tropical Malaria.—Editor.]

Turning to the administration of quinin by the needle, we will consider the following procedures: hypodermoclysis of quinin in high dilutions by normal salt solution, intramuscular injections, Bacelli's intravenous infusions, ordinary hypodermics of quinin, Darker's intracellular injections, injections into the spleen, spinal canal, trachea, subarachnoid, etc.

What I call hypodermoclysis with quinin solutions was first introduced by Grall, and is sometimes spoken of by various writers as the subcutaneous method. The latter term is misleading, and should be employed to designate all hypodermatic procedures. Glatard commends this method, especially in pernicious attacks.

MacGilchrist holds that the method under discussion is not practical and, indeed, says that, "for clinical reasons, subcutaneous injection in extreme dilution cannot, of course, be employed." James, however, takes exception to this statement, and states that "quinin can be given in this way with no more discomfort to the patient than by the hypodermic method, and with far quicker and better results."

The large and uniformly successful experience of Deeks and James with the method and what I have seen of their brilliant results in Panama lead me to believe that in this device we have the best of all procedures yet devised for the treatment of grave malarial attacks. These observers employ the French preparation of a solution of the chlorhydrate of quinin in 0.50-gram (gr. viiss) ampoules, which they dilute with two or three pints of water. A little cocain in ¼ of 1 per cent. solution, injected into the skin at the proposed site of the hypodermoclysis, is found by these authors to prevent effectually any subsequent pain, and they regard the normal salt solution administered to be beneficial in the toxic condition of the patient so treated.

The desirability of high dilutions in this and the following method of exhibiting quinin has been noted by several writers. Giemsa and Schaumann added quinin solutions of different strength to blood serum, and found that the lower dilutions always caused cloudiness, precipitation, and coagulation of the serum, and conclude that similar coagulation and precipitation always follow the injection of low dilutions of quinin into the tissues, and that this fact accounts for the slower absorption. Deaderick states that the rapidity and thoroughness of absorption depend not only upon the solubility of the salt, but also upon "the concentration of the solution. The latter is of the utmost importance, since, no matter how soluble the salt, if given in strong solution, it will not be absorbed."

MacGilchrist in the course of similar experiments went even farther and found that "the salts of quinin most soluble in water, even in a dilution of 1 to 100, are not miscible with blood serum. The three salts commonly used for subcutaneous injection, namely, quinin bihydrochlorate, quinin bihydrobromid, and quinin urea (sic), when prepared in a strength equal to that most frequently employed for hypodermic administration and then mixed with an equal volume of ox-blood serum, produce in a few minutes a solid gelatinous mass. The bihydrobromid salt, even in a dilution of 1 in 20, produces a curdy mass. The deposit which forms *in vitro* when a concentrated solution of a quinin salt is added to blood serum contains quinin in an altered form, oxidized and combined with proteid. When equal volumes of blood serum and quinin hydrochlorid (1 in 20) are mixed together, slightly over one-half of the quinin is dissolved and slightly under one-half remains in the deposit; the greater the dilution of the quinin salts the less the deposit on mixing with blood serum. Quinin salts in the dilutions usually employed are unsuitable for hypodermic use."

This careful observer, from the results obtained through this investigation, forms the following practical conclusions on the subject: "Solutions of quinin salts in the concentration usually employed for hypodermic injection produce, when mixed with blood serum, a coagulum which contains quinin in an altered form. Quinin is precipitated at the site of intramuscular injections as usually carried out. The amount of quinin eliminated in the urine after subcutaneous administration varies between wide limits. Urethane, antipyrin, and sodium chlorid, although they increase the solubility of quinin hydrochlorid in water, do not increase the miscibility of the salt with ox-blood serum; and the new compounds formed in such solutions are of doubtful therapeutic value."

In intramuscular injections I have always used much higher dilutions than were usually designated by writers on the subject and have had large experience with this method. Especially in larger children and adults, and in all severe cases of any age, I have for years advocated these intramuscular injections. In view of my observations in tropical Africa, I am inclined to believe that the numerous accidents arising from this mode of administering quinin are to be attributed to causes other than culpable lack of asepsis on the part of the operator or the effect of the well-diluted salt on the tissues. In the course of many hundreds of injections I have had but one slight abscess, which healed within a few days. These injections include whites and blacks, and young and old.

Until the administration of quinin in very high dilution by hypodermoclysis was brought to my attention I always considered that in extreme cases the only method that could save life was that of intravenous administration, and held that any other method in moribund cases should be counted as nothing less than unjustifiable negligence. James, however, found intravenous injection after the method of Bacelli in dilutions of 1 to 10 gram to 10 c.c. to be dangerous and not satisfactory, and witnessed deaths that followed immediately the injection of quinin according to this method when the dose, dilution, and manner of administration recommended by Bacelli had been carefully observed and embolism could be excluded. According to MacGilchrist this danger may be avoided by great dilution (at least 1 in 150). In my own experience I lost no cases, doubtless because I employed rather high dilutions with salt solution, and I still consider this method an effective one for the treatment of pernicious cases.

Taking up the question of ordinary hypodermics, the low dilutions universally employed have brought the method somewhat into disrepute. In this latter connection, for instance, the brothers Plehn, also Thayer, Laveran, Manson, LeDantec, Craig, and others have brought forward a large mass of evidence to prove the liability of such a procedure being following by nodules, abscesses, necroses, eschars, etc. Deaderick, however, with justice expresses himself on this point as follows: "The necessity of employing a dilute solution has been all but ignored. Nearly all writers on the subject lay great stress on the need of asepsis, but with few exceptions even mention the evils of too concentrated solutions. Strong solutions of quinin injected into the tissues cause a wall of necrosis around the solution, preventing absorption and paralyzing phagocytosis, resulting, even if the solution is sterile, in nodes or ugly chemic sloughs. It is in all probability this chemic irritation of the cells which allows of bacterial infection following the injections of solutions not properly sterilized, and, no matter how sterile the solution, if too concentrated, a nodule or chemic slough is apt to result."

It is only necessary to mention the attempts to inject quinin by needle into the spleen, spinal canal, trachea, subarachnoid space, etc. Such procedures are both unnecessary and unjustifiable, in view of the reliable and effective methods of administration already in our possession.

I have extensively employed rectal injections in the case of children. An important point is that when the salt in solution is placed high up in the bowel the absorption is much more rapid than in ordinary enemata. The salt used should be highly soluble. From twice to three times as much as the usual dose by the mouth can be given *per rectum.* A little tincture of opium (15 drops) may be given with the quinin to quiet the bowel and facilitate retention. Rectal suppositories containing quinin have been used by some authorities, but I have never employed such.

Quinin by inunction, mixed with oil, is spoken of by some writers. I have tried this method with tiny babies, but observed no effect from it. The amount of quinin thus absorbed is probably very small if not actually nil.

Effects of Quinin on the Patient.—Quinin is not strongly toxic in its action on the human organism. Even the kidneys, by which it is largely excreted, suffer but slightly in the process of elimination. In heroic doses it is a heart depressant, and, if sufficient is given, is said to stop the heart in diastole. There are several records of death by quinin poisoning. In one instance 12 grams (ʒiij) and in another half an ounce (15.00 gm.) formed the fatal dose.

Among the most unpleasant effects of quinin is the so-called "quinin fever" seen in tropical Africa. Another is the violent urticaria which sometimes follows administration of the drug. Some individuals seem to have an idiosyncrasy against quinin, and cannot take even small doses.

Contraindications to the Use of Quinin.—In case the effect of the drug is so marked as to endanger the patient, it may be necessary to find some substitute for quinin.

Among distinct contraindications to quinin may be mentioned amblyopia, amaurosis, severe hemorrhages from the mucous membranes, threatened abortion or miscarriage when the mother is not highly malarious, great dyspnea and heart depression, general anaphylactic sensitiveness to the drug (shown by suffocation, urticaria, rise in blood pressure, vomiting, and other usual symptoms of anaphylaxis), and, in some cases, what may be called a hemoglobinuric diathesis in the patient—rendering him, in the words of Koch, a "blackwater fever candidate." In connection with the subject of quinin urticaria Elliott and Bass have pointed out that patients subject to this distressing condition may entirely escape if they remain perfectly quiet in bed during the period over which the effects of the drug extend. The normal effects of the drug, even when rather intense, should not be permitted to constitute a contraindication to its exhibition.

Again, pregnancy should not be allowed to stand as a contraindication to the administration. I have given quinin in moderate doses to many pregnant women without being able to trace a single mishap to the taking of the drug. On the other hand, I have seen many women abort from neglected malarial poisoning. In my opinion, excessive doses are, as a rule, dangerous in such patients, but I have seen badly infected women with threatened premature pains quiet down and go on to term, if quinin was given at the first signal of distress. As Deaderick says, "abortion in these cases is oftener due to too little quinin than to too much."

The matter of so-called "quinin hemoglobinuria" will only be mentioned briefly here, as it will be taken up in detail later under the discussion of blackwater fever.

In the writer's opinion quinin has no specific or even special power of producing hemoglobinuria. It is well known that several drugs will do this in patients who already have a tendency to develop the condition. I remember in this connection one man in West Africa who unwittingly induced hemoglobinuria on several occasions by the use of throat pastilles made up of chlorate of potassium before the explanation of his attacks was suspected. On discontinuing the drug the symptoms disappeared and did not return, although the patient continued to take quinin. Any so-called physiological depression will excite hemoglobinuria in appropriate patients. I have observed the condition to follow chill, prolonged wetting of the body, exposure to sun, fatigue, alcoholic or venereal excess, fits of anger or mental depression, and after no appreciable or untoward circumstances whatsoever, often in individuals who were not taking quinin. Consequently, I do not, as a rule, regard a hemoglobinuric diathesis as a bar to quinin administration. On the other hand, such individuals, in order to escape hemoglobinuria, should have each malarial attack thoroughly treated.

Before leaving the question it should be said that every effort and device should be made to enable the patient to take quinin before resort to substitutes is decided on. Trying different salts may result in success. In some instances when the ordinary preparations of quinin are not borne some of the less toxic preparations, such as Warburg's tincture, euquinin, saloquinin, aristoquinin, etc., may be used with better results.

Again, certain drugs given with quinin may help to obviate its unpleasant effects. Bromids have been highly recommended in this connection, and calcium chlorid is also said to be useful in quinin intolerance.

Substitutes.—If quinin in every form is rejected, methylene blue, arsenic in its various compounds, including atoxyl, iodin, and the iodids, tannin, etc., may be tried. Methylene blue seems to be the most generally approved and popular substitute for quinin. My own experiences with it, however, have not been encouraging, but Ruge, whose opinion is entitled to much respect, considers it as efficacious as quinin. It should certainly be given a trial if quinin cannot be borne.

Arsenic is often given both in its inorganic and organic forms. Arsenious acid in capsules with other tonics, Fowler's solution, and other preparations, all have their vogue in certain quarters. Organic arsenic, atoxyl (monosodium salt of p. aminophenylarsenic acid), soamin (sodium aminophenylarsenate), and even salvarsan (paradiamidodioxyarsenobenzol dihydrochlorid) have been used with varying reports as to their efficiency.

I have employed arsenic, mostly in inorganic form, in large series of cases of malaria and agree with other observers that its value is in the treatment of chronic malaria. In the words of Manson, "the place for arsenic is not as a substitute for quinin during fever, but as a blood restorer after fever."

Various alkaloids of cinchona other than quinin have been advocated by different writers. Among these are cinchodin, cinchonidin, quinidin, and quinoidia. Of such alkaloids cinchonidin seems to be the best, but even this is more toxic and in every way less desirable than quinin.

Picric acid has been recommended in this connection, and in two-grain doses three times a day is said to cause the disappearance of crescents from the blood. I have had no experience with the drug in malaria.

Opium is said to possess antiperiodic value and to be useful in malaria.

Numerous other substitutes have been brought forward by students of malaria, among which may be mentioned splenic extract, eucalyptus, X-ray treatments, salicin, salicylic acid, sodium hyposulphate, various sera, phenocoll, etc. It is only necessary to say of these that none of them appears to be of proved value.

In regard to the last-named drug, which is a derivative of phenacetin, it may be pointed out that, of the laity in the tropics, many seem to have the notion that phenacetin, antipyrin, acetanilid, and other coal tar derivatives are of curative or prophylactic value in malaria. This idea is responsible for many injurious effects following on the regular taking of these drugs. I have seen two cases of collapse supervening on large doses of phenacetin taken for malaria by laymen in West Africa.

Symptomatic Treatment.—In the treatment of symptoms and the general management of cases, the usual methods for the handling of fevers must be employed. Warmth, rest in bed, evacuation of the bowels, diet, and nursing should all be points for care and attention. Hydrotherapy as indicated, liquid food, or at least a light diet, during the attack, and plenty to drink are all recommended. Lemonade or buttermilk are usually grateful to the patient. For headache an ice cap is useful, cold sponging and alcohol rubs are comforting during the pyrexia. Vomiting, if not controlled by other measures, may require morphin. Heart weakness is best controlled by strychnin or brandy.

Treatment of Special Conditions in Malaria.—Under this head we will take up some of the most important special phases of malaria as follows:

Pernicious Malaria.—In this condition, whether the symptoms be that of convulsion, cholera, hyperpyrexia, coma, delirium, collapse, or what not, the first thing to be thought of is to get a sufficient amount of quinin into the circulation in the shortest possible time.

One of the best ways of recognizing a dangerous infection is by estimating the percentage of erythrocytes invaded by parasites. James states that he looks upon an infection of 200,000 parasites per c. mm., or one to twenty erythrocytes, with suspicion, especially if doubly infected erythrocytes are present in the proportion of 5 per cent. or more of the total of the infected cells. I have seen a patient with 30 per cent. of infected erythrocytes and numerous double infections recover after intravenous injections of quinin, but such cases often die even if quinin is administered promptly.

Quinin by hypodermoclysis, intravenously or intramuscularly, in the high dilutions already described is the proper treatment of pernicious malaria. In addition choleraic cases should receive morphin. Hyperpyrexia should be combated by cold baths, and algid cases should be placed in warm blankets with hot water bottles, or other means of producing external heat should be applied. Symptoms of collapse should be combated as described in the preceding pages and in other general works. Quinin by the needle in these cases should be given in full doses. For instance, Grall and Glatard recommend from 10 to 30 grains (0.6 to 2.0 gm.), and James and Deeks give from 20 to 30 (1.3 to 2.0 gm.), even up to 45 grains (3.0 gm.), by this method.

Chronic Malaria and Malarial Cachexia.—These conditions should be treated by quinin in regular doses, and by blood-making and general upbuilding tonic and hygienic measures. Removal from endemic locality is, of course, to be desired when possible. The eradicative treatment of chronic malaria should be sharply distinguished from the attempts to secure and promote latency, which are all that can be hoped for when the patient is obliged to remain in the heavily infected area where he contracted the disease. The prophylactic and latency-promoting use of quinin will be taken up again later under preventive measures. In the present connection it need only be said that thorough quinin treatment by the mouth, followed by the German method of giving 15 grains (1.0 gm.) each

Saturday and Sunday for a couple of months thereafter, is probably as good as any, although most English writers prefer daily doses. Many writers prefer small daily doses following the initial cinchonization. Arsenic in the form of Fowler's solution, or Blaud's pill, and iron, are also to be recommended. The Italian formula of quinin—bihydrochlorate 1.0 gram (gr. xv), arsenious acid 0.001 gram (gr. 1/20), and citrate of iron 0.15 gram (gr. ij)—is a good one. This may be given in the form of pills three times a day—two pills forming an adult dose and one pill the dose for children over three years old. The classical quinin, iron, and arsenic pill used in America is also valuable. Care must be used to ascertain if the pills are soluble and are not being passed unchanged by the patient.

The diet should be nutritious and easily digested. My own experience has confirmed me in the advocacy of high protein feeding for malarial cachectics, and to this I usually add cod liver oil in cases where much weight has been lost.

Warmth, fresh air, sunshine, and the many other obvious hygienic measures valuable in this and similar conditions need only to be mentioned.

Enlarged Spleen.—The treatment of enlarged spleens in malaria needs but a word. In addition to vigorous antimalarial and general treatment some local measures are advisable in many cases. These usually consist of the application of blisters or other counterirritants over the region of the spleen.

I have usually painted the skin with strong tincture of iodin. In some cases I have used an ointment of the red iodid of mercury applied in the presence of artificial heat. Various other counterirritants have been recommended.

Malaria in Children.—Malaria in children deserves a special word. In the writer's opinion too small doses are usually given to children. A good rule would seem to be to give 1 grain (0.065 gm.) for each year of age three times a day. This should be calculated up to ten years of age, above which adult doses may be given if no special contraindications exist. The general treatment is like that of adults.

Conditions Demanding Surgical Interference.—Various conditions due to malaria, or connected with it, demand surgical interference. Among these may be mentioned rupture of the spleen, abscess of the spleen, and malarial ulcers.

Rupture of the spleen when diagnosed calls for immediate laparotomy. When the abdomen is opened "the choice of operation between splenectomy and splenotomy must be based upon individual conditions." Abscess of the spleen is also a surgical condition. While of grave import and frequently fatal, yet these may respond fully to surgical treatment. I have reported successful operation in this condition by Manson's method for liver abscess, using a large trocar and subsequently draining with a single large tube, which was gradually shortened as granulations appeared and the cavity closed. Details of the technique are without the scope of the present work. General treatment, including quinin in full doses, must be carried out at the same time. "Malarial sores" are usually small, definite, round, punched-out ulcers which heal quickly under the local application of any soluble salt of quinin. I have never seen them outside of intensely malarious districts in tropical Africa, although Osler has reported such. General treatment should be given with the local applications.

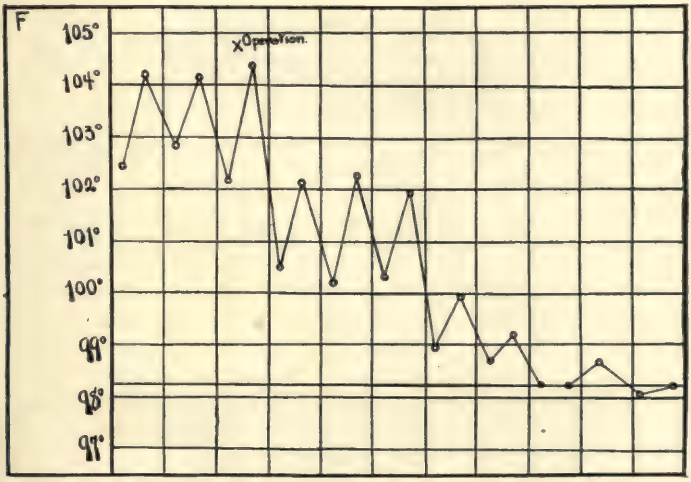

Fig. 4.—Temperature Chart of a Case of Malarial Abscess of the Spleen. Operation, recovery. (After Wellman.)

Convalescence.—Coming finally to the treatment of convalescence, the general rules laid down under hygienic and dietetic treatment must be followed. The quinin and tonics should be kept up, the diet should be generous, and exercise, especially outdoors, should be taken by the patient. He should be kept warm and avoid chills, overexertion, worry, reinfection, and excesses of any kind. A change of climate, attention to the appetite, and occasional laxatives are all recommended. No one should return to a malarial climate until his health is completely restored.

Preventive Measures

The factors involved in the prophylaxis of malaria are many and varied as regards the details involved, but few and simple as regards the general principles concerned.

The first principle has been stated clearly and admirably by Gorgas in the following words: "It has been proved that malaria is carried from person to person by the bite of the female of a particular . . . mosquito, namely, the Anopheles; and we hold that it is conveyed in practically no other way. Our sanitary measures are, therefore, directed toward the destruction of this . . . mosquito. The female Anopheles lays her eggs (about 100 at a time) on the surface of fresh water in which grass and algæ are abundant. These eggs float around until hatched by the sun's heat, and the young larvæ flee to the grass and algæ for protection from small fish and other natural enemies. After larval and pupal stages in the water lasting about eight days, they develop full-grown mosquitoes. The adult Anopheles is weak in flight and does not habitually move about much; probably one hundred yards is practically its ordinary flight. The adult avoids the wind and seeks for its protection undergrowth, grass, and plants near the ground.

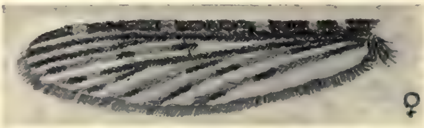

Fig. 5.—Wing of Cellia Squamosa, a West African Anopheline Mosquito. (After Wellman.)

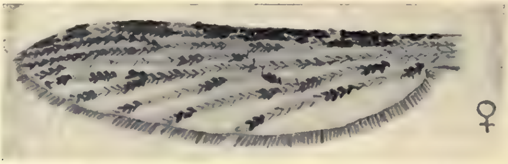

Fig. 6.—Wing of Cellia Pharœnsis, an Angolan Anopheline Mosquito. (After Wellman.)

"Our measures for mosquito destruction are, then, based on the peculiarities just described. We destroy all the breeding places within one hundred yards of the locality we desire to protect, so that the mosquito can find no water in which to lay her eggs, or in which her larvæ can develop. At the same time we clear off the brush within the same area, so that the adult cannot secure protection against the wind."

If we add to this the facts that people when out of protected areas should be defended against the bites of mosquitoes, that infected individuals should be avoided, that quinin should be given to those who become infected, and that the human race needs to be educated along these lines, we have all that is of importance in malarial prophylaxis.

The whole problem is seen to be a biological one and different authors have studied the fish (top minnows, sunfish, sticklebacks, etc.), insects, and other natural enemies of mosquitoes in the hope of finding a way of exterminating these pests.

Sambon has even gone so far as to advance the theory of a hyperparasitism friendly to mankind. Assuming that the "brown spores" of Ross are protozoa (*Nosema*) which feed on the malarial zygotes in the mosquitoes, he points out the possibility of such aid in eradicating malaria from the world.

Among the actual enemies of mosquitoes I have noted in Africa small fish and aquatic Rhynchota preying upon the larvæ and have seen fungi and mites parasitic on imagines. I have also observed a mantis and several species of dragon flies catch them, and have found adult mosquitoes in the crops of nocturnal birds (goat suckers) and in the stomach of a small bat. Whether or not indigenous or introduced natural enemies of African mosquitoes can be utilized in their destruction, however, remains to be seen.

Before leaving this point I ought to refer to the planting of trees, *Eucalyptus*, etc., which has been recommended and practiced by some as a prophylactic measure. I undertook several years ago some experiments as to the effects on the habits of mosquitoes of different plants in favor, and am in accord with other observers that such are valueless in reducing the insects. On the other hand, large plants in the tropics, when cultivated near dwellings, afford shade and even breeding places, and so actually increase the number of mosquitoes. Bananas, palms and large Bromeliaceæ are especially undesirable as affording collections of water suitable for breeding mosquitoes, and these and similar plants are frequently cultivated as being useful or ornamental. In view of my observations, elsewhere published, I agree that the most obvious protection to a dwelling is a large surrounding open space kept clear of high grass and free from depressions that will hold collections of water, since, as has been repeatedly shown, tall grasses, weeds and bushes, by furnishing shade and protection, will lead mosquitoes from their native streams or swamps to houses built some distance away.

Coming now to the practical details which must be attended to if malaria is to be stamped out in any locality, we may mention the following: (1) Destruction of mosquito-breeding places by draining or otherwise lowering the ground water, by filling in of puddles, by emptying receptacles, oiling or poisoning of undrainable water, flooding of intractable areas, etc. (2) Destruction or closure of all protection for adult mosquitoes. (3) Protection of individuals by screening, avoidance of infected individuals, cinchonization, etc. (4) Racial advance along hygienic and educational lines.

Such methods have proven highly successful in the hands of skilled experts. Such instances as Panama and Ismaila are illustrative of what may be attained. On a smaller scale, and with very little cost, much can be accomplished if the desire to be free of malaria be present. In this connection I may say that at one station in West Africa sanitation was carried out under my advice with a view to reducing the endemic malaria of the place. Excavations from which clay for building purposes had been taken were filled up or drained, trees and bushes near the station were cleared away, an irrigation ditch was as well guarded as possible, and the natives were required to avoid accumulation of water near their houses. At the same time quinin was administered freely, not only for the treatment of acute cases, but also in mild cases and as a prophylactic systematically to children who were found to have parasites in their blood, even when apparently well. By these means, carried on over one year, the number of fevers on the station was cut down over one-third. At another station, where the conditions were more favorable, similar methods were used with even better results. At the end of the first year the fever curve at this latter station was shown to be but a little over half of what it had been the previous year, and the succeeding year (1905), in spite of an irregular season, the malarial cases were still further cut down.

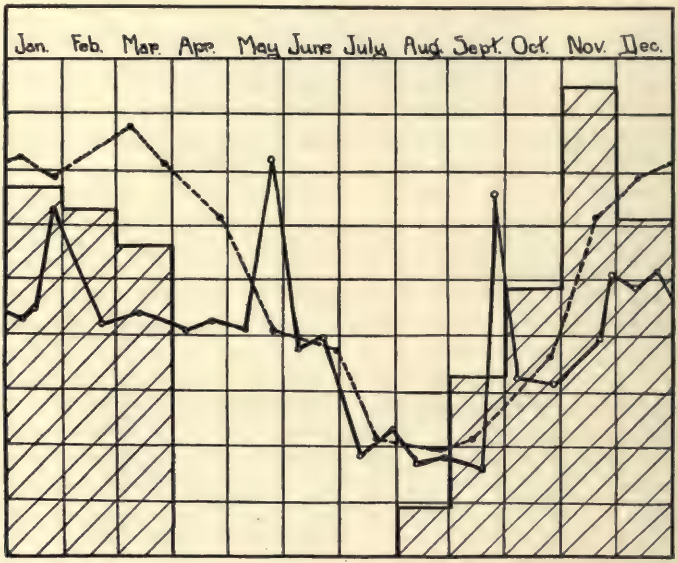

Fig. 7.—Chart Showing the Relation Between the Amount of Rainfall, Number of Anopheline Mosquitoes Taken, and Percentage of Malarial Fevers Among the Natives of One Station in West Africa for One Year. (After Wellman.) The shading indicates rainfall, the dotted line indicates number of anophelines taken from a given amount of wall space in certain houses, the continuous line indicates the comparative number of malarial fevers among the natives.

Note: The epidemics of fever in January, May, and September are probably to be accounted for by sudden changes in the weather, stirring up latent malaria in the natives.

Discussing some of these measures more in detail, we may say, first of all, in the words of Gorgas, that "drainage is, par excellence, *the* means for Anopheles destruction." In such operations use may be made of open ditches, subsoil tiling, "absorbing wells," etc. Of these subsoil tiling is the best, indeed, "the ideal antimalarial drainage system." Open ditches, when intended for such sanitary purposes, must be kept clear of vegetation. In the tropics this means cleaning out twice a month, and, therefore, concrete ditches are much to be preferred in such countries, and even these should be swept out frequently. "Absorbing wells" are useful when the underlying swamps are thin and impervious to water. Holes are bored through this impervious stratum, allowing the water to drain through.

Filling in of low ground when possible is a valuable means of drainage, and, when none of the above methods is practicable, the area may be flooded. In such a case the edges of the water must be kept free from grass and algæ. In this connection may be mentioned rice planting and its relation to malaria. It has been found that rice fields which are permanently flooded are much less dangerous than those in which intermittent flooding is permitted.

In places where stagnant or slow-running water cannot be gotten rid of larvacides must be used. Many substances have been tried, but, of all so far studied, petroleum in some form seems to be the most generally effective. When impossible to drain I have used petroleum in West Africa and have always found it effective in destroying larvæ, pupæ, and often adult females which had come to the water to oviposit. At Panama many experiments were made with various substances, and a larvacide composed of crude carbolic acid, resin, and caustic soda has also been found to have excellent diffusing and poisonous properties, at the same time being an effective germicide. It kills larvæ in dilution of 1:15,000.

The destruction of algæ in water is accomplished by means of copper sulphate. This is inimical to the growth of filamentous algæ, even when used in very high dilutions up to 1:500,000, and will destroy such algæ in dilutions of 1:50,000.

In addition to such measures as the above, all small puddles in the vicinity of habitations must be filled in or periodically swept out. Receptacles that will hold water must be screened or emptied after rains, and, if valueless, broken or buried. Such receptacles as wheelbarrows should be overturned before leaving for any length of time. Sagging gutter pipes must be straightened or pierced with small holes so the water will gradually run out after a rain.

The preceding measures should be carried out for a distance of at least a hundred yards from all habitations, and three or four hundred yards is better. In fact, recent observations on the flight of Anopheles mosquitoes tend to show that our usual estimates are too low. Simpson, for instance, places the occasional flight of females at one-fourth mile, and the brothers Sergent at something less than a mile.

An important point is that trees, brush, and grass should be kept cut between the mosquito-breeding places and the areas to be protected. A few shade trees and ornamental shrubs may be allowed around houses providing the intervening spaces are kept clear.

Coming now to the houses themselves, it may be said that all dwellings in tropical countries should be screened. The size of mesh used in screening is an important factor in the efficiency of this device. Darling found that, out of several hundred mosquitoes, eight common varieties were able to make their escape through a No. 16 mesh wire screen. Consequently, a No. 18 or 20 mesh is necessary for protection.

In spite of every precaution, however, it is rarely possible to rid completely a district of malarial-bearing mosquitoes, and the personal protection of the individual becomes a desideratum. With Europeans a mosquito curtain properly made and properly used is an indispensable precaution. By these means alone I have kept several whites in tropical Africa three or four years after they had had severe attacks of pernicious malaria. In malarious districts the careful use of mosquito net, together with the prophylactic use of quinin, is of great value, even though no general antimalarial measures are carried out. The following table from Celli, whose experience is enormous, illustrates this:

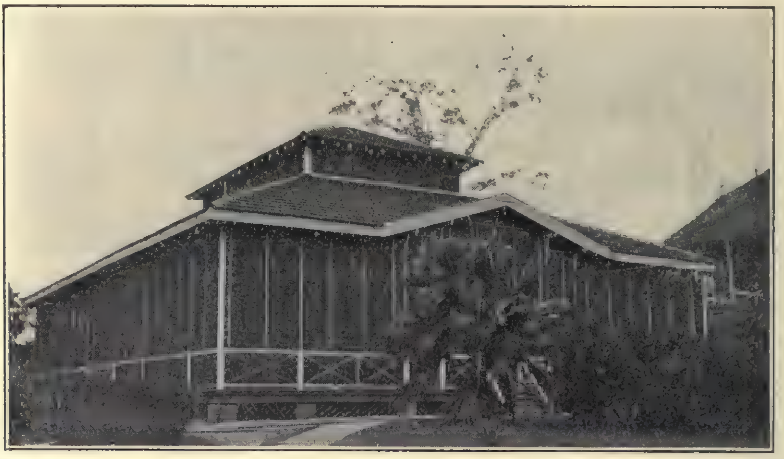

Fig. 8.—Example of Properly Screened Building in the Tropics. Ancon, Panama. (Original.)

Mosquito protection plus quinin	1.75	per cent. of infections
Mosquito protection alone	2.5	per cent. of infections
Quinin prophylaxis alone	20.	per cent. of infections
No protection at all	33.	per cent. of infections

The mosquito net should be rectangular and not dome-shaped, and larger than the bed. The bed should be wide enough so the sleeper does not lie against the net, which should be long enough so it can be tucked under the mattress. The net should be suspended *inside* the framework supporting it, as it is impossible to tuck it closely around a post. It should never be allowed to hang, and in the day time should be folded or twisted so no mosquitoes can get inside. It should be kept free from holes, and when retiring one should get into bed quickly and be sure no mosquitoes have entered with him. All mosquitoes found inside the net ought to be killed.

When sitting in the evening the neck and ankles, especially of women, should be protected by light scarf and high shoes. Electric fans or punkahs are useful in driving away from one's person any stray mosquitoes that may have gotten in the house.

Such substances as oil of pennyroyal, oil of citronella, oil of lavender, etc., are of little value in driving away mosquitoes, as their effect is only temporary and they should never be depended upon for protection.

Occasional fumigation is valuable in destroying mosquitoes that might otherwise escape detection in a screened house. Pyrethrum powder, when burned in a closed room, is effective in stupefying the insects, but they must be swept up and burned afterward, or they will revive. The burning of sulphur is the most effective, as it kills the insects at once. Two pounds of sulphur for each thousand cubic feet of space is the amount needed. Hydrocyanic acid is effective but dangerous. Many other culicides are recommended, but have no advantage over those just named.

It should be pointed out here that avoidance of infected natives is of great importance in escaping malaria in tropical countries. Native quarters should not be allowed near the residences of whites, and native servants should not be allowed to sleep in or near the houses of Europeans.

Drug prophylaxis is an auxiliary method subsidiary to mosquito destruction and protection against mosquito bites, but is, nevertheless, of great value, and in some countries is all that can be done for the native population under present conditions.

The notion that quinin taking is dangerous in tropical countries is a mischievous one, and is productive of great harm. Gorgas, whose opportunities for observation and great authority no one will deny, has said impressively in this connection: "I have seen no evil results that could fairly be attributed to the administration of quinin, and I have often wondered if it could be the same disease which the English have on the west coast of Africa and in which so many of their leading practitioners think that quinin does harm."

Celli, too, speaking of the districts and months of malaria in Italy, says that the prophylactic use of quinin is "as necessary as daily bread," and quinin distribution, as carried out by the Indian, Italian, Greek, and other governments, has resulted in most brilliant success. The following table given by Ross from statistics of the work in Italy is significant:

1895, no quinin distributed	16,464 deaths
1902, 2,200 kilos distributed	9,908 deaths
1909, 23,635 kilos distributed	3,463 deaths

The immediate object of quinin prophylaxis is to place the system in such a condition by the administration of the drug that "the development of symptoms after a first or fresh infection is inhibited" (Brown). The remoter objects are avoidance of relapses and preventing the victim from becoming a menace to his fellow men. The prophylactic use of quinin is also considered by many writers to ward off pernicious attacks and to render any fevers contracted more amenable to the therapeutic use of the drug.

Craig has well pointed out that the vigorous treatment of initial cases and the discovery and proper treatment of latent infections are points of great importance.

The first of these is not sufficiently understood by too many medical men in the tropics. All frank cases should be promptly and thoroughly treated along the lines already laid down in this article. Even slight attacks should never be treated lightly. As Ziemann has insisted, real quinin prophylaxis consists in killing the parasites which invade the body at the beginning of their development and even before fever has set in. Hence, promptness and thoroughness of treatment must always be insisted upon. It should never be forgotten that gametes will develop in 9 to 15 days, and that small doses of quinin stimulate the patient to the production of these forms so potentially dangerous to those living in contact with him. When symptoms show themselves it must be remembered that large doses of quinin are both a preventive measure against pernicious types of malaria and a guarantee of the patient's future welfare in a malarious country.

The discovery and treatment of latent cases are not only important to the individuals personally benefited, but also to their neighbors, as it has been shown that about 50 per cent. of all cases of latent malaria are gamete carriers.

The number of latent cases of malaria in endemic regions differs according to the age and other circumstances of the inhabitants. I consider the ascertaining of the spleen index a useful method of determining the percentages of severe infections and have used this method in West Africa.

The following table from Craig shows the latent infection at various ages in a large number of cases from several malarious regions of the world:

Age in Years.	No. Examined	No. Infected	Per Cent. of Infections
1-5	1,684	502	29.8
5-10	1,645	463	28.1
10-15	1,390	437	31.4
Adults	4,931	1,139	23.0

The presence of so many gamete carriers in tropical countries always constitutes a grave menace to the rest of the population, and systematic quinin administration is of the greatest value in ridding a locality of such a handicap. While in West Africa I consistently advocated to the laity that native children, who without much harm to themselves harbor the parasites in their blood in great numbers, should be systematically dosed with quinin.

Isolation or at least screening at night of gamete carriers is also to be recommended.

The effect of quinin administration on the gametes, causing these to disappear from the peripheral circulation in about 8 days, is a valuable weapon in our hands. The drug prevention of gamete formation has already been mentioned.

The exact method of attaining the ends agreed upon has been much in dispute among the advocates of quinin prophylaxis in malaria, and two schools of opinion have grown up around the subject. In general it may be said that the British, French, Italian, and American observers are in favor of small daily doses (3 to 6 grains—0.2 to 0.4 gm.) of the drug, while the German investigators, following Robert Koch, recommend larger doses (15 grains—1.0 gm.) on 2 or 3 successive days at 8 or 10-day intervals.

Various modifications of these schemes have also been proposed, among which Ziemann's method of giving large doses at 4-day intervals deserves special mention. His program is as follows: (1) 1 gram (gr. xv) quinin every 4 days; (2) 1 gram (gr. xv) euquinin every 4 days if 1 gram (gr. xv) quinin is not tolerated; (3) ½ gram (gr. viiss) quinin every 4 days if 1 gram (gr. xv) euquinin is not tolerated; (4) ½ gram (gr. viiss) euquinin every 4 days if ½ gram (gr. viiss) quinin is not tolerated. He suggests as a general rule to take the dose on all dates divisible by 4 up to the 28th, and then to begin again on the first of each month.

Taking up the comparative values of the so-called English and German methods, the recent Uganda experiments are of interest. The Europeans were divided into the following classes: (1) Those taking 5 grains (0.3 gm.) daily; (2) those taking 10 grains (0.6 gm.) every Wednesday and Saturday; (3) those taking 15 grains (1.0 gm.) every 8th and 9th day; (4) those taking quinin irregularly; (5) those taking quinin only when sick from fever. The result of this investigation was interpreted as indicating that system 1 is the best, and 2 and 3 the worst, of those mentioned, but the series were hardly large enough to be conclusive.

From my own experience in tropical Africa I am inclined to the German mode of prophylactic administration, and would sum up my own opinion by saying that, as a rule, the most that can be hoped for in the case of many whites who are careless regarding the instructions of their medical adviser is continued latency. This can best be secured by actively treating each malarial attack, afterward administering to an adult smaller doses of quinin at the average rate of a grain an hour for one week. Subsequent to this may be given on two successive days in each week a fifteen-grain (1.0-gm.) dose, keeping this up for a period of two months. I also recommend taking about thirty grains (2.0 gm.) a week in two doses during the most trying part of the wet season.

Before leaving the subject it may be pointed out that, while the most soluble salts are, as a rule, best for treatment, many writers consider that the least soluble salts are the best for prophylaxis. On this theory the nucleinate and tannate are recommended, the latter in the form of chocolates for children. In view of recent researches, however, little difference should be expected when the drug is administered by the mouth.

The prophylaxis of malaria through general hygienic measures—in other words, by increasing human resistance through good food, good houses and clothing, and sound hygienic *régime*—is worthy of mention and not to be neglected, while more specific measures are being carried out for the individual and the community.

Education of the public in these matters by state boards of health, through addresses by members of the medical profession, through the daily press, and in our public schools, is a great need. The establishment of a National Commission for the Study and Prevention of Malaria, as Harris has suggested, would result in a great advance toward the final conquest of this scourge.

If the public could be taught to realize that the expense of freedom from malaria and other mosquito-borne diseases at Panama is only 9 mills per day per capita, and that Italy by free quinin and other measures has made a death spot livable, much could be done by us as a nation for our southern states and for our tropical neighbors and dependents.

BLACKWATER FEVER

This is one of the gravest diseases of Europeans in Africa and other tropical countries. In this disease more than in most the success of treatment depends upon prompt and careful attention to a number of details.

Without pronouncing upon the nature of the supposed relation, which is denied by many, between blackwater fever and malaria, I will say that my investigations in West Africa led me to the conclusion, long held by many others, that they are related in some way. This relation, however, may be only accidental and geographical.

I have shown that the principal malaria-bearing mosquitoes, severe endemic malaria, and blackwater fever closely coincide in their geographical distribution in the part of tropical Africa studied by me; also that cases of blackwater fever examined at an early stage of the disease can usually be shown to have malarial parasites in their blood. Even later examinations, as a rule, show pigmented leukocytes, or an undue percentage of large mononuclear leukocytes. In these observations my experience agrees with that of Christophers and Stephens, Deeks and James, and others.

It is claimed by some that the parasites are always present, but that the vitiated erythrocytes containing plasmodia are always the first to be destroyed, with their contained parasites, when the terrific hemolysis sets in. This is possibly true, but too much cannot be made of this argument, as it leaves other factors unexplained. For instance, it does not account for the absence of crescents in the blood. One expects to find gametes in the blood of old neglected cases who oftenest develop blackwater fever, for the gamete is a rough weather form calculated to stand hard usage, so to speak, in its passage through the varied conditions in the mosquito's stomach, which must very greatly differ from its former habitat in the human blood stream. So that it is hard to think of any subtle change in the serum of the blood that would destroy it. This and other considerations, which cannot be discussed here, would lead one to believe that the character of the relation between the two diseases has not yet been explained.

An hypothesis which the writer is now investigating and which was suggested to him by a series of observations on patients, both living and *post mortem,* which observations are not yet complete, is the following: that blackwater fever is due to a distinct species of malarial parasite, morphologically related to the ordinary estivoautumnal parasite, from which it has not yet been specifically distinguished, but which further study will show to be a good species and etiologically related to this disease. The establishment of such a doctrine would go far to account for several inexplicable features of blackwater fever, including its epidemiology, distribution, and relation to other fevers. It is my intention to carry on these investigations as material and opportunity offer.

Other etiological theories, such as the absence of chlorids and excess of sulphates in the blood serum, decreased alkalinity of the blood, etc., need not be discussed in an article like the present.

In the course of the disease the kidneys often, but not always, show some edema and frequently nephritis. The spleen is enlarged, dark, sometimes necrotic, much as in any profound toxemia. The bone marrow is almost always hyperplastic. The liver is usually enlarged, sometimes with focal necroses. The gall bladder is full of a thick, coagulated, or finally granular black bile, the brain is usually pale.

The four cardinal symptoms of the disease may be said to be fever, hemoglobinuria, jaundice, and vomiting. There is sometimes pain in the head, occasionally pain in the epigastrium. The liver is often tender, but the kidneys are usually not so. In the later stages a systolic hemic murmur may be heard. The urine is acid, containing albumin, and full of dark granular detritus. Casts and epithelial cells are present. Blood cells are usually absent. The hemoglobin in the blood may fall to thirty per cent.

and recovery still take place. Some patients are almost acorpuscular before death.

The most usual complications are tonsillitis, *otitis media,* abortion and miscarriages, and purpura. Relapses frequently occur. Among the sequelæ are cachexia and, more rarely, chronic nephritis.

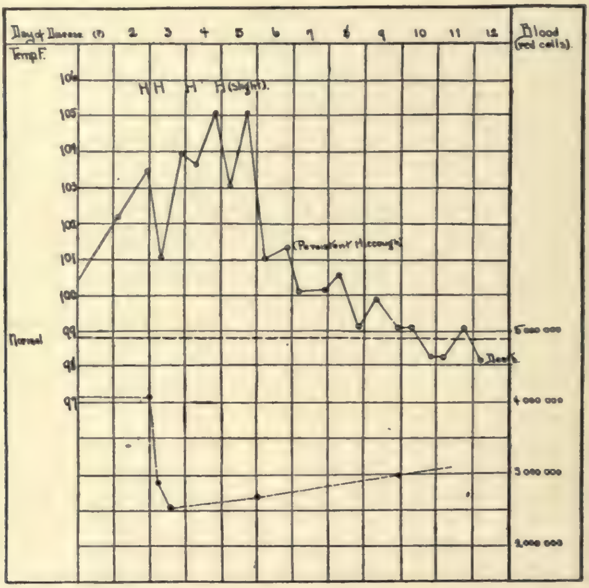

FIG. 9.—COMBINED BLOOD AND TEMPERATURE CHART OF A FATAL CASE OF BLACKWATER FEVER IN WEST AFRICA. (After Wellman.) The dotted line indicates the number of erythrocytes per c. mm.; the continuous line, the temperature in the mouth. H- hemoglobinuria.

Remedial Measures

The patient should at once be put to bed, and absolute quiet and rest enjoined. He should not be allowed to lift his head unaided. An initial dose of calomel, 3 to 5 grains (0.2 to 0.3 gm.), in my opinion, exerts a beneficial influence on the subsequent course of the disease, although several very recent writers dispute this. The patient should be kept warm.

When urine becomes scanty hot fomentations should be applied to the region of the kidneys. Large amounts of fluid must be gotten into the patient. If nausea and vomiting prevent him from taking them by the mouth, they should be given as enemas in the form of Murphy drip, injected into the subcutaneous tissues of the flank with a large Pravaz syringe, or introduced intravenously. Normal salt solution should be employed in the last named of these procedures. When enemas or the Murphy drip are employed, not more than 1/5 of 1 per cent. of sodium chlorid should be added to the water, so that absorption will take place rapidly.

The question of quinin administration in blackwater fever is a vexed one, and those who have not had experience with large series of cases should not hastily offer opinions. Mannaberg, Marchiafava and Bignami, Bastianelli, Thayer, Vedy, Craig, Deeks and James, Deaderick, and others have all discussed the question at great length. Probably the statement that will carry the agreement of the greatest number of observers can be given in the words of the last-named author as follows: "The only conditions in which quinin is indicated are, first, where the parasites show no tendency to disappear after forty-eight hours from the onset; second, in the infrequent cases of intermittent hemoglobinuria, where the outbreak corresponds with parasitic sporulation."

When quinin is given it should, as a rule, be administered by needle, as in severe malaria. All drugs, however, are useless in the great majority of cases. The patient's strength must be kept up by liquid nourishment at frequent intervals either by the stomach or rectum. The best stimulant for the heart weakness is strychnin. Some authors recommend brandy.

After the attack is over the patient should have a good course of tonics, fresh meat, and other nourishing food, with wine, if advisable.

As for the symptomatic treatment, the vomiting and restlessness are best controlled by morphin hypodermically and crushed ice. In heart failure, where brandy or other stimulants by mouth are not absorbed, strychnin may be given hypodermically.

I have often thought of treating patients in the earliest stages of the disease with serum obtained from immune natives living in severe endemic blackwater districts, but have never carried out the plan.

Widal has thought that the hemolysis of paroxysmal hemoglobinuria is due to the lack of an antiamboceptor which is present in the blood of normal persons, and has tried as a remedy serum of a rabbit which had been injected with human blood, and believed that the effect was good.

Recently Fischer has recommended in cases of nephritis intravenous injection of an alkaline salt solution (see under Cholera). I have had no experience with this treatment, but on general principles should consider it rational. [Since this hypertonic solution produces some hemolysis when injected intravenously, it would appear safer to administer it *per rectum* in blackwater fever.—Editor.]

Preventive Measures

According to the writer's experience with the disease in West Africa, there are two classes of patients. The first can endure almost any number of attacks. One hears of men who have suffered fifteen or twenty attacks, or even more, but this class is unfortunately so small as almost to remain out of the account when deciding whether a given patient dare remain in the tropics or not. The other class, which in Africa includes the vast majority of cases, can, under ordinary treatment, usually endure one or even two attacks, but often succumb to the third or fourth. Roughly speaking, the danger increases in geometrical ratio with the number of attacks. So, at least in West Africa, it is of the first importance to take into account the number of previous attacks. The occasional very slight attacks, lasting only a few hours, which are sometimes sandwiched in between the grave attacks, are not a constant enough feature seriously to affect calculations. The writer's rule in Africa has been to send the invalid home after each of the first attacks and to refuse to pass a man to go out after the second.

It is of great importance in persons who have had one attack of blackwater fever that each malarial attack be thoroughly treated. It may be said that the prophylaxis of blackwater fever is really the prophylaxis of malaria. In tropical Africa, especially, there is a widespread idea among the laity that the prophylactic use of quinin is certain to end in an attack of blackwater fever. Countless harm has been done by the advocates of the theory that quinin is alone responsible for hemoglobinuric fever. As a matter of fact, I have never in eight years' experience in the tropics seen a case of blackwater fever in a patient who had taken quinin properly as a prophylactic measure, and Koch himself, whose authority is cited by the advocates of the quinin poisoning theory, believed and taught quinin prophylaxis not only against malaria in its ordinary forms, but also against blackwater fever itself.

3. TRYPANOSOMIASES

Creighton Wellman

Trypanosome infection in man is exceedingly common throughout tropical Africa, the local distribution depending upon the presence of tsetse fly areas. The writer had opportunity for thorough study of the disease in Portuguese West Africa, where it is not uncommon. Some of the cases showed practically no symptoms even while the trypanosomes were abundant in their circulation for long periods. One case, watched for about two years, from an apparently healthy condition went through the entire gamut of trypanosome fever into typical sleeping sickness. The disease is of enormous economic importance. In South America there is also a disease caused by infection with a similar parasite.

A. Trypanosome Fever and Sleeping Sickness

This disease is caused by infection with the flagellate protozoan, *Trypanosoma gambiense,* a flagellate protozoan which measures about

15 μ-30 μ by 5 μ-2.5 μ. The flagellum rises from the blepharoplast and is continued along the side of the parasite, attached to it by means of an undulating membrane, and is prolonged into a free flagellum. The nucleus is ovoid and is situated slightly behind the center of the parasite. As yet the parasite has not been cultivated on artificial media, but this will probably be possible with Bass's newly described method.

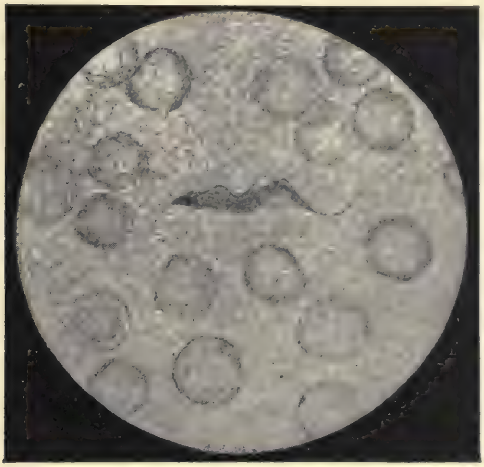

FIG. 10.—BLOOD FROM CASE OF SLEEPING SICKNESS. (Original.)

Another slightly different form of the parasite is *T. rhodesiense,* which is probably a distinct species or at least a variety.

The conditions set up by the parasites' presence in the body are various. In typical cases the skin is harsh and dry, there is a papular eruption somewhat resembling recently acquired scabies. The tongue is usually coated, pointed, and tremulous. I have usually found the spleen to be enlarged, although this may be from coincident malaria. There are often deep hyperesthesia and enlargement of the cervical and supraclavicular glands. Anemia, edema, headache, irregular fever, sometimes loss of weight, drowsiness, torpor, tremors, and convulsions are also seen. In some cases the temperature may be subnormal. There may be profuse diarrhea. In some cases loss of sexual power, amenorrhea, iritis, delirium, and other nervous manifestations have been reported.

The pathology of the condition shows it to be a meningoencephalitis. There are congestion of the meninges, effusion of lymph, turbid cerebrospinal fluid, and small-celled infiltration of perivascular lymphatic tissue throughout the brain and other parts of the body. The pathology resembles that of general paralysis of the insane.

The causal organism is disseminated by one of the species of tsetse flies, *Glossina palpalis.* My own observations on the habits of these flies were obtained from the study of a distinct geographical race of the species in question, which has been described by Austen under the name of *Glossina palpalis wellmani.*

The normal food of these flies is human blood. The flies hide in the tall grass and sedges near rivers and streams, and also on stones, trunks of trees and vines, and among the leaves of shrubs and bushes on the bank. The flies do not always remain close to streams. I have seen them between three and four hundred yards from water in thin desert bush, which affords practically no shade. When a native ventures into the fly belt the flies rise from their resting places seeking for an opportunity to bite. On several different occasions I followed natives going to a river to fetch water. One of these I saw bitten twice, three were bitten once each, and seven I did not see bitten at all. Many of the specimens I took also had abdomens greatly distended with blood.

Glossina palpalis wellmani at least does not share the dislike for human ordure which has been popularly ascribed to the other tsetse flies. I have frequently seen it in and around the filthiest native camps at some distance from rivers, where it had evidently gone for the purpose of sucking human blood. According to my observations and the observations of many others, the fly bites most viciously during the heat of the day and less readily during the evening and early morning. The natives claim that it occasionally bites at night, but my experiments do not bear out this statement. I have not observed that heavy rains reduce the numbers of flies, as has been reported by others.

Remedial Measures

The problem of the cure of trypanosomiasis in man is a complicated one and many efforts have been made to find a substance that will kill the parasites and yet not unduly injure the host.

It has been found that one substance alone will not do this, but Ehrlich has conceived the idea of removing chemoresistance by changing the trypanocidal substances employed in the treatment. He argues syphilis and some forms of malaria as analogous problems to be thus met.

So the most hopeful treatment has come to be that of combined therapy, and the employment of any trypanocide alone has ceased to be considered justifiable, since even full doses, for instance, of organic arsenic will not kill all the trypanosomes in the body, and full doses of such substances are accompanied with grave danger. With combined treatment smaller doses of each contributing remedy may be employed, resulting in a united and more effective result.

Another principle is that of alternations in the agents administered on account of the fact that the parasites become habituated, as it were, to one trypanocidal substance, and cannot be eradicated even by heroic treatment with it, but in their weakened state another substance may finish them and complete the cure.

The substances which have been shown to be of most value may be divided into five groups as follows: (1) Arsenical compounds, (a) organic, (b) inorganic; (2) antimonial compounds; (3) benzidin compounds and analogous dyes (trypan-red, etc.); (4) basic triphenylmethane dyes (parafuchsin, etc.); (5) mercurials.

1. Arsenical Compounds.—Atoxyl.—Coming at once to the question of specific treatment, we may say that, of the preparations of arsenic, atoxyl has, on the whole, given the most favorable results to date. This substance, which is the monosodium salt of p. aminophenylarsenic acid, has been shown to exert a specific action upon the trypanosomes, which in a few hours (four to five) become sluggish and altered in appearance, the leukocytes meanwhile becoming noticeably increased in numbers. After seven or eight hours the parasites begin to disappear from the blood, the leukocytosis steadily increasing, and phagocytosis of the trypanosomes having been observed.

The exact mode of its action is not yet clear, but it is held by some to be through the formation of what is known as trypanotoxyl, an hypothetical compound of a reduction of atoxyl with albumin, and which must be formed before atoxyl can act upon the trypanosomes. Other writers hold that the action of the drug is, more probably, directly upon the parasites.

As to the matter of dosage, two types of opinion obtain among workers in this field. One school holds that large doses should be given at first before the trypanosomes become resistant to the drug. On the other hand, Manson and others believe that such drugs should be used in small doses and over long periods.

Observation of the cases treated by me in West Africa inclines me to the belief that Manson's opinion in this regard is sound.

It may be said in general that not more than 0.5 of a gram (gr. viiss) should be given at a dose under ordinary circumstances, and that it should be employed in as high a dilution as is practicable. A 20 per cent. solution is often recommended, but several workers have complained, in personal communications to the present writer, of the practical difficulty of getting over a 10 to 11 per cent. solution. Uhlenhuth, in speaking of the strength of solutions, says that "*cæteris paribus* the weaker are better borne." It should be pointed out here, while discussing dosage, that the effects of the drug, when given by the mouth, seem to be more toxic than when it is administered by the needle, and consequently that larger doses by the mouth than by the needle cannot be given. In cases where 0.4-0.5 gram (gr. vi-viiss) can be given intramuscularly or intravenously, not more than 0.5 gram (gr. viiss) can be given by the mouth without danger.

As regards the time of administration, it may be said that the consensus of opinion seems to be that the earlier the treatment is begun the greater the chance for cure. The explanation of this is probably that the arsenic does not penetrate, or at least only feebly penetrates, the meninges, and consequently that cases treated before the invasion of the subarachnoid space are curable, while the rest are not. Another point that is insisted upon by Ehrlich and others is that, when possible, the atoxyl should be given while the trypanosomes are numerous in the blood, as it is then the chances of killing them are the greatest.

Mode of Administration.—Taking up the question in more detail, it may be said that the drug has been given by the mouth, hypodermically (intramuscularly and intravenously), by inunction, and intraspinally. Intravenous injections with proper precautions are the best method of exhibition, although many observers recommend ordinary subcutaneous administration. In this connection I may quote Moore and Nierenstein, who say that, "by medical men who will take the necessary precautions, atoxyl is best given intravenously." It may be added that the drug is thus best exhibited in a normal saline solution. That oral administration gives rise to symptoms of poisoning more rapidly than any of the subcutaneous methods, and that high dilutions are preferable to concentrated ones, has already been pointed out.

Contraindications.—When the toxic symptoms of atoxyl are noticed the drug should be discontinued, at least for a time, and some substitute employed. The symptoms of poisoning to be noted are: dryness of throat, headache, giddiness, faintness, fever, colic, vomiting, diarrhea, nephritis, retention of urine, strangury, deafness, disturbances of vision, paresis, abdominal cramps, and slowing of pulse. Atoxyl amblyopia deserves a special word, and at its first onset the drug should be immediately stopped.

SUBSTITUTES FOR ATOXYL.—Before leaving the subject of atoxyl, mention must be made of some of the atoxyl derivatives. Various of these have been used when atoxyl itself was not well borne. Among these acetylated atoxyl and its sodium salt, and also the paroxybenzylidene derivative, have been used. All of these, especially the sodium salt (called arsacetin), are much less toxic than atoxyl, although arsacetin cannot be given to human cases in large doses.

Another compound which has been used with apparent success is sodium aminophenylarsenate, known as soamin. This preparation has been employed by some observers with apparently as good a result as atoxyl, with none of the poisonous effects of the latter drug. It appears to differ from atoxyl only in the amount of water in combination. Ten grains (0.6 gm.) every alternate day for twenty days may be given.

Arsenophenylglycin, especially, has very recently attracted attention as a valuable substitute for atoxyl. Strong and Teague, for example, write that "arsenophenylglycin has proved to be by far the most satisfactory means of treatment of trypanosomiasis yet discovered." While very efficacious in animals, even more effective than salvarsan, yet, on account of the hypersensitiveness to this drug which is so often developed in man, it must be regarded as a very dangerous substance to use as a routine treatment.

Salvarsan itself has been employed, but has not been found so valuable as in spirochetal infections.

Other organic arsenical preparations, such as orsudan, are being investigated by different workers, and may still be held *sub judice.*

Inorganic Arsenical Preparations.—Inorganic arsenic in the form of orpiment, arrhenal, sodium cacodylate, and arsenate of iron has also been employed hypodermically, either alternating with atoxyl or as a substitute for it. Fowler's and Donovan's solutions have also been similarly used, both hypodermically and by the mouth, and arsenious acid has been used intramuscularly.

2. Antimonial Compounds.—The sodiotartrate of antimony (Plimmer's salt) has been employed in human trypanosomiasis, notably by Manson. As much as two grains (0.3 gm.) per dose have been administered. Hypodermically it causes pain and even sloughing, and is, therefore, best given by the mouth with plenty of water. Tartar emetic and Schlippe's salt (sulphid of soda and antimony) have both been used intravenously.

Anilin antimonyl tartrate and different antimony compounds of thioglycollic acid, e. g., sodium antimony thioglycollate, have been the subject of considerable experimental work, and are being used both alone and as alternates to arsenical substances and other drugs. Probably the most that can be said of antimony in the treatment of human trypanosomiasis is "that it ought to form an adjunct to arsenic, and in certain cases might replace it" (Broden and Rodhain).

3. Benzidin Dyes.—Trypan-red and trypan-blue have both been used with some success in the treatment of trypanosomiasis of animals, and it has been thought that they should be also of service in the treatment of human trypanosomiasis. Other similar color groups, among which may be mentioned tryparosan, are at present being tried in this connection.

4. Basic Triphenylmethane Dyes.—Parafuchsin is well borne in full doses and seems to have distinct preventive properties, and, associated with organic arsenic, should be of value, and deserves careful and more extended trial.

As in the case of antimony, the principal value of the various dyes mentioned will probably prove to be their effects when used in combination and alternation with arsenic.

5. Mercurials.—Bichlorid of mercury has been used intravenously, but has not seemed to be of much benefit. It has been thought that mercury or its salts should prove a valuable adjuvant to atoxyl on the theory that salts of heavy metals would attack the stage of the trypanosomes which is not amenable to arsenic. Opinion is much divided as to the results obtained by mercurials either alone or combined with arsenic.

Other Remedies.—A large number of other remedies have been employed by various observers, among which are iron, quinin, methylene blue, thyroid extract, chrocoidin, etc. Quite recently experiments have been carried on with both p. and m. aminophenylstibinic acid, with no very encouraging results, however. Serum therapy has been attempted with experimental animals, and also in man, but so far with no definite advance in our control of the disease. The use of horse serum was attempted early in the history of sleeping sickness investigations, but serious symptoms, probably due to anaphylaxis, have followed upon its administration. Finally, nothing practical has come out of the attempts to employ ultraviolet and other rays in the treatment of trypanosomiasis.

General Treatment.—The general treatment of the disease is most briefly summed up in the words of Manson, who remarks that "every effort should be made to keep the patient in good general health and thereby increase his resistance. Rest, warmth, and residence in a cool and healthy climate, the systematic use of quinin for any concurrent malarial infection, and the avoidance of fatigue and everything that might depress should be insisted on." Of course, such measures are impossible when dealing with savages, whose miserable surroundings can rarely be immediately ameliorated.

Surgical Treatment.—Excision of the large cervical lymph nodes in the early stages of sleeping sickness has been practiced by native Africans and recommended by some European practitioners and writers. Such a procedure seems of doubtful value.

Convalescence.—In cases where apparent cure is achieved, great care during convalescence should be employed to prevent any depressing or otherwise trying factors from making the patient a chronic nervous invalid.

Preventive Measures

The prophylaxis of human trypanosomiasis may be said to depend upon the proper handling of three factors: trypanosome, tsetse fly, and man, both infected and healthy. The methods adopted must, therefore, aim to attack the fly, to protect healthy people, and to prevent infected persons from becoming a menace to their fellows. Among the measures employed, clearing of fly belts, segregation and treatment of the sick, removal of people residing in fly areas, and instruction of natives are the most important.

The following recommendations, made by me in 1905 looking toward the prevention of the spread of trypanosomiasis in a district in West Africa, are illustrative of what may be aimed at in drafting preventive regulations:

(1) Native camps should be removed to points some distance from rivers and kept there by police. Of course, this, as a rule, meets with some native opposition; but half a dozen native soldiers stationed at such camping places would, as a rule, be sufficient to enforce such a regulation. The natives can be induced to carry water for cooking and drinking purposes some distance, as is proved by the location of many established camping places where the water supply is sometimes situated nearly a mile from the camps.

(2) Squads of soldiers, laborers, and other controllable bodies of men should be passed through the fly belts at night.

(3) Native quarters in cities, towns, villages, especially visited by native caravans from the interior or coast, should be moved as fast as possible from points on rivers to the hills farther away.

(4) The government should not permit sleeping sickness cases and suspected cases of trypanosome fever brought from surrounding endemic districts or from other parts of Africa to be landed at the port or ports of the district to be guarded.

(5) Care should be taken not to allow recognizable cases which already exist in the district to be transported either from the coast to the interior or, vice versa, through the fly belts.

Such regulations, or similar ones, when properly carried out are of considerable influence in controlling the spread of the disease.

Among more general measures, clearing of fly areas from all brush and tall grasses or weeds has been found invaluable. For a distance of fifty yards or more from infected streams all vegetation, excepting short grass, should be cleared, and this clearing should extend up and down stream at least a hundred yards beyond the limits of the fly belt. In some districts, where the flies are very abundant, much greater areas than this should be cleared, and Wölfel found that in some districts cleared areas of even 200 or 300 meters were not enough to keep them entirely off the road. The cultivation of lemon grass (*Citronella citratus*) on the site of old fly belts has been recommended, as has also the planting of *Eucalyptus* trees, these both being obnoxious to tsetses.

Another important measure is the abandoning of all fly areas. It is recommended that European houses be moved at least half a mile from the water, and, if possible, to higher ground. Natives should not be moved to points near their old haunts. They should be taken far enough away so that they cannot easily return. Cases of trypanosomiasis especially should be promptly removed from inhabited fly belts. One infected person may infect and destroy a whole village or neighborhood. Clandestine labor recruiting in Africa complicates the problem of keeping infected natives out of healthy areas. The scheme of deportation and segregation into camps where the natives are treated and prevented from returning to infected fly belts, as tried, for instance, in Uganda, has not proved successful enough to give great hope of thus stamping out sleeping sickness.

The ignorance and superstition of the native black throughout tropical Africa are formidable obstacles to any comprehensive plan of prophylaxis, as he dreads the possibility of being sent to a camp, and, in the words of Barclay, "will go to any length to avoid either himself or his relatives being segregated."

In addition to avoidance of natives, the avoidance of close proximity to animals should be recommended in trypanosomiasis districts. Parasites of the *Trypanosoma gambiense* type have been found in monkeys, and other animals may likewise be "sleeping sickness reservoirs."

Trypanosomes of various types have been found in horses and cattle, buffaloes, sixteen species of antelopes, hyenas, pigs, etc. Antelopes are easily infected with *T. gambiense* and should at least be considered as "potential reservoirs" (Bruce).

Coming now to the discussion of essentially personal precautions, it may be said that in traveling one should avoid camping in infested areas. Camps should be set at least half a mile from the edge of a fly belt. In case it is necessary to cross streams inhabited by "fly," this should be done at night, or at least early in the morning or late in the evening. When unavoidably detained in a fly belt one should take every care to avoid being bitten. The danger is especially great when fishing.

It may be pointed out here that it is very important to be able to recognize tsetse flies and to be able to distinguish them from other flies. In the absence of such knowledge the novice may unwittingly establish his camp in a deadly area. According to Austen, our best authority, with whom all African travelers will agree, "It is probable that only those who have suffered from the attacks of tsetse can recognize them on the wing, but in the resting position their identification is easy. In this attitude they can be distinguished from all other blood-sucking diptera, especially from those belonging to the genera *Stomoxys* and *Hæmatopota,* which are most likely to be mistaken for them, by the fact that the brownish wings lie closed flat over one another down the back, like the blades of a pair of scissors, while the proboscis (i. e., the proboscis ensheathed in the palpi) projects horizontally in front of the head."

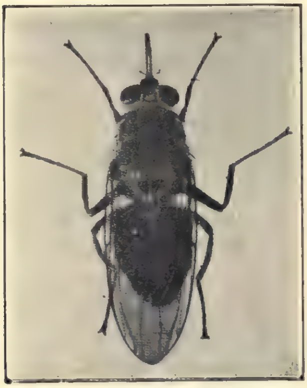

FIG. 11.—GLOSSINA PALPALIS. (After Austen.)

The above is true of all tsetse flies, of which there are some ten species. *Glossina palpalis,* the carrier of sleeping sickness, is of a deeper brown than the others, so that "persons familiar with tsetse flies recognize *palpalis* at once by its darker coloration."

A number of tsetsefuges have been devised by different authors. Manson suggests naphthalin and camphor. Eucalyptus oil and citronella oil will either of them keep off tsetse flies for about an hour, and are of value when unavoidably detained in a fly belt.

It is said that the upper decks of vessels are much freer from "fly" than the lower decks.

Women, especially, should wear leggings or puttees when in fly areas. The tsetse fly loves to get into dark and protected places, e. g., under a petticoat, and different cases of sleeping sickness in white women give a history of being bitten on the ankles.

In addition to the more general measures against the fly which have already been discussed, there has been devised by Maldonado a device for trapping adult tsetses. This is done by placing on the backs of plantation laborers a black cloth on which is plastered bird lime made from the latex of *Ficus, Loranthus,* and other plants. The flies are attracted by dark colors, and, descending to bite, are caught in large enough numbers to diminish sensibly their attacks.

It must not be forgotten, while on this subject, that there are natural enemies of the tsetse, among which may be mentioned drought, birds (drongos, bee-eaters, fly-catchers, swallows, stonechats, etc.), lizards, shrews, and various insects, e. g., wolf flies (Asilidæ). Possibly some natural enemy might be imported or cultivated that would eradicate the flies.

Preventive inoculations with dead trypanosomes have been tried as a prophylactic measure, but without visible result.

Drug prophylaxis has been studied with some attention. The treating of cases having the parasites in their blood is, of course, the most important measure. Orpiment has been recommended as a prophylactic for persons exposed, and arsenophenylglycin has been shown to be a good prophylactic in horses and cattle (Schilling). The problem of securing exposed persons against the disease by means of drug administration is a difficult one, and its solution problematical.

The matter of education is an important one in the prevention of human trypanosomiasis. The prophylaxis of sleeping sickness should be carefully and simply explained to chiefs and other intelligent natives. Missionaries and others who know the native dialects can be of great service to the indigenes in this respect; for, in order to carry out the best regulations against the disease, the coöperation of the native peoples must be elicited. One of the greatest difficulties at present is their apathy and indolence. As Hodges says: "They seem to expect to be paid by the government for any labor which they are recommended to undertake to save themselves from extermination."

In conclusion I may sum up the things to be done in the following rules, compiled from the Sleeping Sickness Bulletins: 1, Clear fly-infested areas; 2, fill up or drain pools, etc.; 3, cultivate plants noxious to fly (*Citronella, Eucalyptus*); 4, destroy the animals on which fly feeds; 5, encourage natural enemies of fly; 6, collect or destroy pupæ or adult flies; 7, remove villages or markets to fly-free areas; 8, discourage or forbid occupations carried on in fly belts (fishing, etc.); 9, avoid fly belts in heat of day; 10, select roads which do not traverse fly belts; 11, protect the person by suitable clothing, nets, screening, etc.; 12, use tsetsefuges when unavoidably in fly belts; 13, instruct natives in avoiding fly; 14, segregate the sick away from fly and from healthy persons; 15, prohibit moving of persons from infected to uninfected fly districts; 16, prohibit moving of infected persons from one infected district to another; 17, treat all infected persons.

B. Infantile Cachexia in Brazil

Chagas has described a second form of human trypanosomiasis in South America caused by another flagellate, *Schizotrypanum cruzi.* In this parasite there is an unusually large egg-shaped blepharoplast near the posterior end. The nucleus lies longitudinally and is oval or longitudinal in shape. The flagellum which rises from the blepharoplast has a free portion of variable length. Other forms of the parasite occur occasionally, some of which are intracorpuscular. It may be cultivated on hemoglobin agar.

The chief conditions produced by the presence of the parasite are anemia, degeneration of the parenchymatous organs, and delayed development with infantilism. Edema, enlargement of the glands, spleen, and liver, and various manifestations, including imbecility, are all described.

The parasite is said to be carried by a Reduviid bug, *Conorrhinus megistus.* The disease may be transmitted by the bite of the bug to lower animals.

Remedial Measures

Our scant knowledge of the nature of this parasite and its relation to the disease described by Chagas forbids more than the suggestion that the measures outlined above for the treatment of infection with *T. gambiense* and *T. rhodesiense* are also appropriate in this case. As the disease seems to be one of children, care should be taken in the proper reduction of dosage when using the toxic substances recommended.

Preventive Measures

As it seems clear that the Reduviid bug transmits the parasite the rational prophylaxis would be directed toward protecting persons from its bite. The habits of this bug are similar to those of the bedbug, and cleanliness, disinfection of infested houses and beds, together with the use of a night light when sleeping in questionable quarters, are all indicated.

4. LEISHMANIASES

CREIGHTON WELLMAN

These diseases, long obscure as regards their etiology, have been shown to be due to infection with protozoa belonging to the genus *Leishmania,* and it is not improbable that others, the cause of which is at present unknown, may in time be demonstrated to depend on similar parasites.

A. KALA-AZAR

This is a chronic disease due to infection with *Leishmania donovani,* a flagellate protozoan, closely allied to, if not identical with, the genus *Herpetomonas.* It contains a large nucleus and a smaller blepharoplast. There is a small chromatin rod placed near one edge and a rudimentary flagellum has also been described. The organism may be cultivated in citrated blood. It has two stages of development, an intracorporeal in humans and possibly other mammals, and an extracorporeal which probably takes place in insects.

The parasite appears to invade many different tissues and to be widely distributed in the body, although it seems to attack specially the endothelial cells of the circulatory system. In this habitat the parasites have been obtained from the liver, spleen, kidney, lungs, glands, arachnoid space, blood, and in lesions in the intestines and skin. The parasites are almost entirely found in tissue cells in which they multiply, enlarging and eventually destroying the cells sheltering them. Thus set free, the parasites invade others, the process inducing fever, which is at first more or less recurrent, the exacerbations being marked by splenic and hepatic enlargement. Later the fever and enlargement of the spleen and liver become chronic and are accompanied by sweating, anemia, and wasting, and sometimes by a peculiar pigmentation of the skin, edema, bleeding from the gums and nose, pains in the limbs, etc. The cases seen by the writer in general appearance suggested chronic malarial poisoning. The disease is usually fatal, often through intercurrent affections.

Remedial Measures

The experience of those who have had the best opportunities to observe the effect of drugs has not been encouraging. Early removal from the endemic region has occasionally resulted in apparent cure. One of the cases seen by the writer recovered on being promptly invalided from India to Europe, although the convalescence was a matter of many months and the patient's health remained permanently injured. Quinin, arsenical preparations, salicylic acid, and various other remedies have been advocated in the treatment of the disease.

1. Quinin.—Castellani and Chalmers and also Rogers and Price advocate this drug as a routine treatment. Manson considers the beneficial effect to be due only to the clearing up of the complicating malaria which is usually present. Quinin sulphate, two grams (gr. xxx), and euquinin, two grams (gr. xxx), are daily given by the mouth, with which the authors quoted combined hydrochlorid, one gram (gr. xv), and quinin cacodylate, 0.25 gram (gr. iv), administered intramuscularly on alternate days. Rogers advises heroic amounts, four to six grams (gr. lx to gr. xc) daily until the temperature falls, after which he continues the drug in smaller doses, one gram (gr. xv) daily for some weeks. Recently Muir claims to have reduced the mortality of the disease in a striking manner by using quinin in acid solution to produce a serous effusion and subsequent leukocytosis. He employs the following formula: quinin sulphate, 32 grains (2.0 gm.), dilute sulphuric acid, 1 dram (4 c. c.), distilled water, 4 drams (16 c. c.). From 20 to 90 minims (1.3 gm. to 6.0 gm.) of this solution are injected intramuscularly. It causes great pain, and cocain must be previously injected. As the swelling subsides the injection is repeated. Craig, on theoretical grounds, suggests that the intravenous injection of quinin would be the most efficacious way of administering it.

2. Arsenic.—Atoxyl was the preparation tried experimentally by Manson and Low, and it was administered by intramuscular injection of two grains (0.2 gm.) every day or every other day, according to the patient's tolerance.

In my opinion, if given at all, the drug should be pushed to its physiological limit or up to 0.65 gram (gr. x) per day.

The results are not all that might be expected in view of the effects of the drug on the closely allied parasitic flagellates, and Manson himself is not enthusiastic on the point. The usual precautions must be observed in the use of this drug.

Soamin is claimed to have benefited some cases, and this might be tried in instances where the more toxic preparation is contraindicated. Possibly the derivative of atoxyl known as arsenophenylglycin might be likewise substituted instead of the original atoxyl. Various other arsenical preparations have been suggested by different authors.

3. Other Remedies.—The salicylates, different preparations of antimony, fuchsin, senega, thymol, phagocytin, the antistaphylococcus serum advised by Rogers, and bone marrow have not yielded noteworthy results. Prolonged counterirritation over the splenic region is recommended by

Indian observers. Good food, nursing, warmth, and rest in bed are, of course, all indicated.

Among the complications, troubles of the digestive tract are most common; therefore, the diet should be bland and easily digested. Diarrhea should be treated with milk diet and intestinal antiseptics or bismuth preparations, as symptoms indicate. Dysentery should be treated with massive doses of bismuth subnitrate with astringent enemata if there are ulcers low down in the large intestine. The various intercurrent affections, tuberculosis, pneumonia, etc., should be treated according to approved methods in such instances.

Preventive Measures

The cases should be considered as infectious and dealt with accordingly. In view of Patton's researches bedbugs and biting Reduviidæ should be destroyed, patients sick with Kala-azar isolated, and well persons in endemic areas should avoid being bitten by vermin. Infected quarters should be burned, and the clothing and furniture of infected persons disinfected.

B. INFANTILE SPLENIC ANEMIA

This disease, distributed over North Africa, Sicily, and Italy, is caused by a second species of *Leishmania,* also morphologically indistinguishable from *L. donovani,* which has been named *L. infantum.* The affection, as its name indicates, seems specially to affect children.

The pathology and symptoms of infantile splenic anemia are very similar to those of Kala-azar, the effects upon the spleen, liver, etc., and the severe anemia being prompt manifestations.

Remedial Measures

The treatment is no more satisfactory than in the case of Kala-azar, and no specific has been discovered. The same drugs used in Kala-azar in doses suitable to the age of the patient should be tried. The experiments of Nicolle and others with electromercurol, colloidal electrargol, and colloidal thiarsol have added nothing to our methods of treatment. Röntgen rays have likewise been tried without much benefit.

Preventive Measures

According to Nicolle, dogs are dangerous in endemic localities, as they are carriers of the infective parasite. Vermin should be destroyed or their bites avoided, and cases of the disease should be isolated and fomites burnt or disinfected.

C. ORIENTAL SORE

This distressing affection is due to *Leishmania tropica,* morphologically indistinguishable from *L. donovani,* and is distributed over North Africa, Asia Minor, parts of the Mediterranean, India, Turkestan, etc., and probably in South America.

The symptoms are both local and constitutional. The characteristic ulcers are infiltrated, especially at the edges, with large mononuclear cells which contain vast numbers of the *Leishmania.* The process is essentially a chronic one, and, while the sore and its resulting scar are troublesome and unsightly, yet little danger is to be feared from the disease, and spontaneous recovery is the rule. The object of the treatment should be to shorten the disease and to prevent mutilation and secondary infections.

Remedial Measures

The best treatment of oriental sore is thorough cleansing with some non-irritating antiseptic solution and the application of a bland ointment, either of boracic acid or zinc oxid. Destruction of the papule or ulcer by caustics or cauterization at any stage is liable to result in a flaring up and extension of the process.

A large number of local applications have been advised by different observers, among which may be mentioned a 2-4 per cent. solution of sulphate of copper, touching with nitrate of silver, at intervals of three or four days, washing and dressing, meanwhile, with a potassium permanganate dressing, curettage, cleansing with 1:1,000 solution of bichlorid, followed by an antiseptic ointment, exposure to X-rays, 5 per cent. permanganate ointment, covering the sores with sheet lead, etc. Billet has lately recommended methylene blue as a local application. Gueyat advises thorough cleansing and application of the following ointment:

Salicylate of methyl	5 grams	(gr. lxxv)
Salicylate of bismuth	2 grams	(gr. xxx)
Subnitrate of bismuth	1 gram	(gr. xv)
Oxid of zinc	20 grams	(ʒv)
Glycerin	10 grams	(ʒiiss)

General treatment should be instituted when the patient's condition is, as is often the case, anemic and debilitated. Iron tonics, eradication of malaria, of intestinal worms, with other obvious measures, should be attended to. Change of climate, when possible, and avoidance of reinfection from the patient's own sores or from others are important. Salvarsan has been given by Nicolle with partial improvement. In spite of all endeavors, the disease may drag on for months and even years.

Preventive Measures

One of the most interesting facts regarding oriental sore is that an immunity can be established. Protective inoculation has been practiced by the Jews of Bagdad, much as inoculation against smallpox was in vogue before the discovery of vaccinia. As oriental sore is a disease of countries where the camel is used as a beast of burden, and camel drivers are invariably infected, it is probable that young camels or some other animal might be infected with the human strain, and a vaccine produced which would confer an immunity to the severe type of the disease. Avoidance of infected animals is, of course, indicated as a preventive measure. The well should also avoid touching infected persons or handling objects which have been in contact with patients. In view of the probable transmission of the germ through the bite of insects, the bites of all arthropods, such as flies, mosquitoes, *Simulium, Phlebotomus,* etc., should be guarded against; and, when such bites are incurred, they should be cauterized or otherwise disinfected. All wounds infected while in endemic areas should be similarly treated.

5. HISTOPLASMOSIS

Creighton Wellman

Possibly related to the foregoing conditions is a disease reported by Darling in Panama and considered by him to be due to a protozoan parasite related to *Leishmania,* and described under the name *Histoplasma capsulatum.*

Calkins seems in doubt regarding the parasitic nature of Darling's bodies, and it is rather significant that no cases since the original ones have been reported. The lesions illustrated in Darling's papers are hardly conclusive without further material.

6. TROPICAL RELAPSING FEVERS

Creighton Wellman

Spirochetal fever in the tropics is widely distributed. In view of the fact that the parasites are usually very scarce in the blood, they are likely to be overlooked unless the examination is very complete. There seem to be at least two great groups of tropical spirochetoses, one of which is peculiar to tropical Africa and the other to the oriental region. A third group, as yet not well studied, is probably peculiar to tropical America.

The form of the disease best known to me is the so-called tick fever of tropical Africa. The disease is caused by the presence in the blood of a spirochete (*S. duttoni*) distinct from the causal organism (*S. recurrentis*) of relapsing fever in temperate climates. This parasite measures about 16 μ in length, being nearly twice as long as *S. recurrentis.* In dried specimens it has a tendency to an arrangement in circles or double circles. The writer was the first to point out that *S. duttoni* as seen in stained specimens dies in rounder and more flowing curves than *S. recurrentis,* a character adopted later by Novy in his separation of the two species.

The presence of the parasite in the blood had, in a case carefully studied by me, a constant relation to the rise and fall of the temperature and accompanying symptoms, and I was able to show that the clinical picture conforms, in some instances at least, to typical cases of relapsing fever at home.

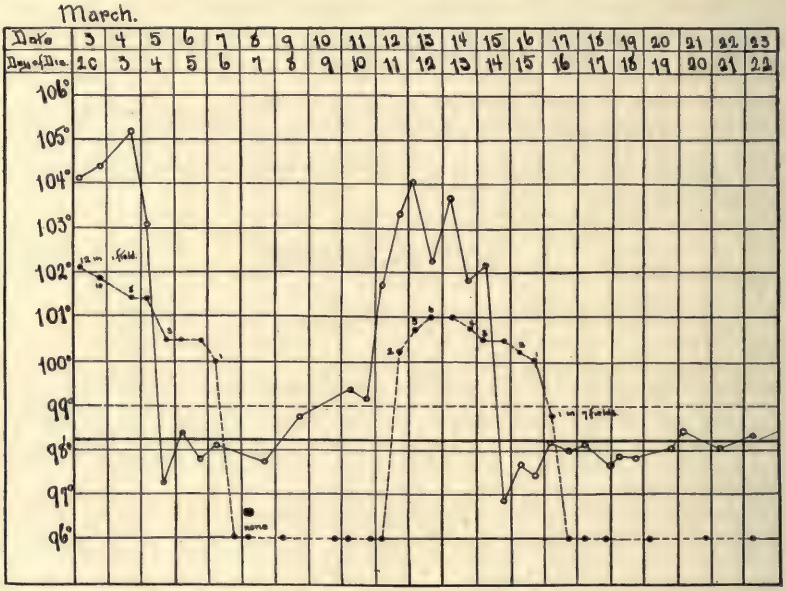

FIG. 12.—COMBINED BLOOD AND TEMPERATURE CHART OF A CASE OF AFRICAN TICK FEVER. (After Wellman.) The temperature is represented, in the usual manner, by an unbroken line. The dotted line represents the comparative abundance of the parasites in the blood during different stages of the attacks, taking the line marked 100° as representing the average of one parasite in each field. Thus, 101° would mean 6 parasites in each field; 102°, eleven parasites. This case was remarkable for the great number of spirochetes in the blood, as these are usually very scarce.

Balfour has recently described a new species (*S. berberi*) as the cause of the relapsing fever of Algeria. *Spirochæta carteri*, the cause of the Indian fever, is distinguished from *S. recurrentis* by being thinner and forming less regular spirals.

The parasites are carried by arthropods, in case of the African fever by a tick (*Ornithodoros moubata*), and in case of the Indian fever by bedbugs. The Persian disease called miana fever is probably also a spirochetosis transmitted by another tick, *Argas persicus*. The tropical American form, which has very regular spirals, is known as *Spirochæta novyi*.

The African tick fever is sometimes very severe and not seldom fatal. An interesting fact in connection with the ticks which carry the African disease is that they are at times attacked by a fungus which destroys them. The disease generally begins at one edge of the tick's body in the form of a light, almost white, spot, which spreads. The infected ticks are more sluggish than the healthy ones. These ticks are a veritable pest throughout the whole of Portuguese West Africa. They infest the huts and camps of the natives in great numbers. I have found as many as a hundred in an hour in an old native hut. In some places they become an intolerable plague, and the blacks are not seldom compelled to burn their old kraals and build new ones in order to escape them. They hide, like the bedbug, in the thatch and in the cracks of shelters and houses, and come out at night to bite. They bite both man and all domestic animals. I have found as many in pigsties as in any other one situation. Like the bedbug, this tick will not bite except in the dark.

REMEDIAL MEASURES

The general treatment is much like that of typhoid fever. In view of the fact that immunity can be established in this disease, the possibilities of serum treatment are to be borne in mind. The serum of immune animals causes *in vitro* rapid degenerations of the parasites; Lowenthal and Dobrosrakow have both used the sera of horses immunized to the parasites of relapsing fever with apparently some good results.

As was to be expected, diamidoarsenobenzol hydrochlorid (salvarsan) has proved of the greatest use in the treatment of spirochetal fevers, and has revolutionized the treatment, which before its introduction had been entirely symptomatic. Iversen employed 1 per cent. aqueous solutions, and from 0.05 to 0.4 gram was injected intramuscularly. He treated fifty-two patients. Forty-one of these were treated during the first attack, four of them by intravenous injection. Eleven patients were injected during the second attack. In all these cases the fever ceased by crisis and the spirochetes disappeared from the blood. The Yakimoffs report similar success with animals inoculated with *S. duttoni*.

Besides salvarsan, other organic arsenicals have been successfully used in cases of human tick fever. Among these are atoxyl, arsacetin, arsenophenylglycin, arsenophenol, atoxylate of mercury, etc. Salmon has reported moderate success with antimony, and various analin colors and benzidin dyes have also been investigated in this connection. Of all these salvarsan seems to be the most effective.

For the pains in the joints and limbs morphin is indicated. The temperature is best controlled by the application of cold. Vomiting should be combated by the use of cracked ice. In case of collapse heart stimulants must be employed. The nursing, diet, and general management of the case are of the utmost importance.

PREVENTIVE MEASURES

The following recommendations made by the writer in West Africa illustrate principles which are applicable to the control of the disease wherever it may occur or whatever be its disseminator.

(1) The tick in question (*O. moubata*) should be regularly destroyed in crowded centers by disinfecting natives' houses, barracks, and other permanent quarters and by burning old camps, huts, etc. As it retires into cracks during the day, replastering is a good device when houses cannot be destroyed.

(2) Soldiers, laborers on plantations, etc., should be made to keep their houses clean and to sleep in hammocks, or on beds well raised from the floor and away from the wall. I was able by changing soldiers, etc., from floor beds to hammocks, the ropes of which were wrapped in cotton (over which the tick cannot crawl) to stamp out a little epidemic in one district. Natives should never be allowed to sleep in or near the quarters of Europeans.

(3) Soldiers, porters, servants, plantation laborers, and other controllable bodies of natives should be compelled to observe regulations regarding regular bathing and washing clothes.

(4) Natural enemies of *O. moubata* might be found and introduced and encouraged. I have reported on a Reduviid bug (*Phonergates bicoloripes*), which feeds principally if not exclusively upon this tick. I also described on one occasion a mold which in some instances destroys them. One can hardly see how the bugs could be utilized in this connection, but experiments might be made with the fungus.

When sleeping in infected houses, as in the case of bed bugs, a night light will prevent the ticks from biting.

The room, furniture, and belongings of patients should be disinfected so that all vermin are surely killed. Bedding can best be disinfected by heat.

7. DISEASES DUE TO OTHER FLAGELLATA AND THE CILIATA

BALANTIDIUM COLI

WILLIAM B. WHERRY

Ulcerative colitis, in which the *Balantidium coli* plays an important primary or secondary part, appears to be widely distributed, cases having been reported from many parts of Europe, from Asia, Africa, North and

South America, and from the Philippine Islands. Strong (3) (1904) collected and summarized the findings in 124 reported cases, including two of his own.

The lesions and symptoms simulate those of intestinal amebiasis. Bowman studied ten cases in Manila. Of these two died. Most of the cases exhibited no symptoms excepting an occasional diarrhea. Balantidia only could be found during these attacks. He suggests that at this time "nests" of the ciliates are set free by ulceration, which perhaps follows the local plugging of blood vessels by the parasites.

The histologic studies of Solowjew, Askanazy, Klimenko, and Strong have demonstrated beyond question the distribution of the parasites in the mucosa, submucosa, and in the blood vessels of the submucosa. However, it is still unsettled whether the Balantidia are capable of penetrating the mucosa in the absence of a preëxisting lesion. The absence of polynuclears in the neighborhood of early lesions would seem to rule out the primary action of bacteria (Bowman). Numerous experimenters have failed to infect dogs, cats, rabbits, guinea-pigs, monkeys, and pigs by the feeding or injection *per rectum* of infected feces.

A Balantidium, which is believed to be identical with that found in man, is found as an apparently harmless commensal in the intestinal tract of the domestic pig. Some believe that under unknown conditions man and other animals acquire their infection from the pig. That some unknown underlying conditions are necessary to prepare a host for infection or infestation may be argued from the failures to infect young healthy pigs.

Brooks (1902) concluded that an outbreak of dysentery among the orang-outangs in the New York Zoological Park was due to *Balantidium coli.* Noc (1908) reported natural infection in a monkey (*Macacus cynomolgus*). Brumpt (2) has reported a case of Balantidium colitis in a *Macacus cynomolgus.* He was able to transmit the disease to six other *macaci* and transferred it from the monkey to the pig and from the pig to the monkey.

From a study of 111 cases reported with sufficient completeness Strong concludes that 32 (29 per cent.) recovered; and that, while the mortality was apparently 30 per cent., a number of these cases died of other diseases. The same writer concludes that the "prognosis seems to depend upon the general condition of the patient, and upon whether he has sufficient strength to undergo local treatment, the extent to which the disease has progressed, and upon the coexistence or non-existence of other diseases."

Treatment

Quinin enemas, given as directed under intestinal amebiasis, seem to have been as successful as any other method of treatment. Success seems to depend upon the patient's ability to take a sufficiently long course of treatment. Vincent has suggested the use of one-half per cent. Labarraque's solution (sodium hypochlorite) given as enemas. The rapid cure of a case treated with ipecac is reported by Duncan.

[The ordinary vermifuges, santonin, filix mas, and santonica itself, have given good results in individual cases. Appendicostomy with irrigation should be considered, as is done in bacillary dysentery.—Editor.]

FRAMBŒSIA TROPICA

PAUL G. WOOLLEY

Frambœsia tropica or yaws is a specific infectious disease very similar to syphilis in its manifestations. It is, as a rule, the result of inoculation at breaks in continuity of the skin, and, like syphilis, is characterized by three stages. It differs from syphilis in that it is the result of invasion of the body by an organism (*Treponema pertenuis,* Castellani), which, though extremely difficult to distinguish from *Treponema pallida,* is nevertheless different. It also differs from syphilis in being autoinoculable, in not being in any sense hereditary, in not presenting lesions of the central nervous system, and in that the cutaneous manifestations are extremely uniform.

In past centuries it is possible that frambesia was not uncommon in temperate zones. Castellani suggests that at one time it was epidemic in Scotland and Ireland. It is also possible that this disease, or one of a very similar nature, exists at the present time in Greece. But, with this one possible exception, frambesia is a disease of tropical and subtropical localities, and, when cases are occasionally seen in other latitudes, they are imported examples. Such cases appear most frequently among soldiers and sailors who have served in infected districts.

The term frambesia was first used by Sauvages (1759) to indicate the resemblance of the typical lesions to the raspberry (Fr. framboise). Charlouis (1882), to whom we owe our first definite knowledge of the relation which exists between frambesia and syphilis, used the designation *polypapilloma tropicum.* In Brazil the term *bubas* or *bobas* is in use.

Very soon after the first description of frambesia, the suspicion was entertained that this disease bore an intimate relation to syphilis; either that it was syphilis modified by a racial immunity which was the result of years or centuries of infection, or that it was the syphilis itself modified by climatic conditions. As early as 1848 Paulet showed that inoculation was possible and that typical lesions developed in from 12 to 20 days after infection, and of this fact Charlouis made use when later he inoculated successfully with syphilis a native who was affected with frambesia, and observed the development of the lesions of both diseases side by side, and showed that the course of neither disease was modified by the other. Still later Neisser in his series of experiments with apes and monkeys corroborated the earlier work of Paulet and Charlouis, and showed that each disease gave rise to an immunity which had no effect upon the course of the other; and, furthermore, he also demonstrated, by means of the Wassermann reaction, that the antigens of frambesia and syphilis are distinct.

In spite of such demonstration, Duprey insists that Castellani has only muddled the etiologic problem in frambesia, and that the infectious nature of the disease has yet to be proved. He believes that a free diet of mangoes predisposes to, and that unhygienic surroundings assist in, its production.

As has already been said, infection occurs at the site of some break in continuity of the cutaneous surface of the body. Such breaks may appear of very minor importance. Thus in children the primary lesions are not infrequently on the elbows and knees, about the mouth, nose, and the other natural body orifices. Similar extragenital primary lesions are not infrequent in exotic syphilis, but, whereas syphilis is not, at least as a rule, autoinoculable, frambesia is, and from the primary lesion infection may be carried by the fingers or clothes to other parts of the body. As in syphilis, so in frambesia, the infection may be carried from person to person in a variety of ways. Clothing, bedding, drinking cups, all play a rôle, some more frequently than others. The common oriental custom of wiping the fingers upon window ledges, door posts, chairs, and in fact any convenient article or object, is surely an important factor in the spread of this and other infectious diseases, especially syphilis and leprosy. The rôle of insects has been promulgated by many and investigated by a few writers. And, as Gudger says, the theory of the rôle of insects, especially of flies, is a very old one. Gudger quotes from the account of Koster's travels in Brazil (1809-1815) as follows: "This horrible disorder is contracted by inhabiting the same room with the patient, and by inoculation; this is effected by means of a small fly, from which every precaution is oftentimes of no avail. . . . If one of them chances to settle in the corner of the eye or mouth, or on the most trifling scratch, it is enough to inoculate with bobas, if the insect comes from a person who labors under the disease." A still earlier account Gudger quotes from Stedman's travels (1796) as follows: "Indeed, if a fly, which has been feeding upon the diseased, lights upon the slightest scratch on a healthy person, it communicates this dreadful disease." Castellani's experiments, while not at all conclusive, hint at the possibility of the intermediary part played by insects. Robertson, by careful attention to bodily cleanliness among yaws patients, and by smearing their sores with mercurial ointment, has prevented the spread of infection among hospital patients, and he suggests that this result has been due to the fact that flies could not become infected by feeding at the lesions. It seems from the collected evidence that, if insects are able to convey the virus, they do it in a mechanical way, and that they are in no sense hosts. And, when we know that the secretion of the frambesial sores contains the treponemas, it should not be remarkable if insects might transmit the organism, provided these are deposited upon open wounds. Still, the fact that in experimental infection it is necessary to scarify deeply and rub the infected material well in seems to indicate that insect transmission is not a common method. It seems that the sore-picking habit is much more important. An oriental native will amuse himself for a long time picking off the dried secretions of his sores, thereby soiling his hands, which he wipes off on whatever object is near. With such filthy hands he fondles his children, feeds them, or picks their sores, and is, therefore, much more able than the flies to spread infection.

Prophylaxis

The manner of transmission of frambesia indicates the prophylactic measures to be employed, and these measures are entirely similar to those to be taken in preventing syphilitic infection. Persons suffering with yaws should be isolated until their lesions are healed, and their clothes as well as the utensils which they have used should be disinfected. Individuals who are subjects of this disease should on no account be allowed to trade in the market places where they can contaminate the food or the dishes of eating places. Personal cleanliness on the part of the uninfected, and especially careful attention to even small cutaneous lesions, will prevent infection to a great extent.

Treatment

Treatment is, as a rule, very satisfactory, and recovery is rapid. Nowadays it seems that Ehrlich's salvarsan will do much to simplify and hasten a cure, especially if it be followed for some time by the older and very satisfactory potassium iodid or mercury, or both.

It has been said that certain individuals do not react well to antisyphilitic treatment, and that many of these react well to decoctum sarsaparillæ compositum.

In my own experience, which has been exclusively with children, potassium iodid has been used for internal medication, and the mercury used only for external and local applications—for bathing and dressing lesions. Iodoform has also been used with satisfactory results, either as a powder or in the form of an ointment.

Arsenic preparations, in the form of Fowler s solution, or atoxyl may often be of great service, particularly the latter, which may approximate in its effects those produced by "606."

For the primary sores little need be done beyond cleansing and protecting them, best by bichlorid dressings, or with calomel, or iodoform powder. Ulcers should be treated antiseptically and, if necessary, surgically. It is possible that in some of these ulcerations, which are quite resistant to treatment, the use of dilute solutions of basic fuchsin suggested by my colleague, Dr. May, will be of value. The eosin treatment has failed in my experience.

In convalescence tonic treatment is desirable.

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CHAPTER V

DISEASES DUE TO TROPICAL CESTODES, TREMATODES, AND NEMATODES

1. TENIASIS

CREIGHTON WELLMAN

Infection with tropical cestodes, strictly speaking, is not a very common condition, although the cosmopolitan tape worms are abundant enough throughout tropical and subtropical countries. The species discussed below may be considered as more or less special to hot countries.

A. SOMATIC TENIASIS

The invasion of the tissues with larval tapeworms is not uncommon in tropical and subtropical countries, and cysticercoid and hydatid diseases are more or less frequently reported, but are not deserving of special description as tropical infections. Three Dibothriocephalid worms, however, should be particularly mentioned here.

(1) *Sparganum mansoni.*—This tapeworm, in the plerocercoid stage, has been reported from China, Japan, and other parts of the far East. The parasites are variable in dimensions, and may measure 8 cm.-36 cm. by 0.1 mm.-12 mm. Similar parasites have been found in east Africa and British Guiana.

(2) *Sparganum proliferum.*—This parasite has been reported from Japan and from the southern part of the United States. It is very variable in size and shape, but may attain 1 mm.-12 mm. by 2.5 mm.

(3) *Sparganum baxteri.*—Measures 15 cm. in length by 1 mm. in breadth. Reported from German East Africa.

These larval tapeworms have been found under the peritoneum in the neighborhood of the kidneys, in the iliac fossæ, in the pleural cavity, in the urethra, beneath the conjunctiva, and in the subcutaneous connective tissue. The definite host has been thought to be probably a carnivorous domestic animal, possibly the dog, cat, or pig. Others have conjectured that the carrier is more probably an aquatic bird or fish. Tumors, abscess, etc., have been found to be due to the presence of the parasite.

When the worms invade the urinary passages symptoms of stricture, pain, and hematuria are described. In cases of infection with *Sparganum proliferum* the parasites are found encysted in the subcutaneous tissue in large numbers. In the cases reported a history of previous intestinal teniasis was elicited. *S. baxteri* was found in an abscess in the thigh of a Masai.

Remedial Measures

The treatment of this condition is essentially surgical. When the presence of *Sparganum mansoni* causes excrescences on the surface of the body, these should be removed by operation. When the cavities of the body are invaded, resulting in alarming symptoms, surgical relief should be sought if the cause of the disturbance can be diagnosed. As for the treatment of infection with *S. proliferum*, the cysts containing the parasite may be easily enucleated, the relief, as in the case of *S. mansoni* infection, being sought in surgical measures.

Preventive Measures

Definite prophylactic measures cannot be outlined. In view of the probable method of transmission, and arguing from analogy, avoidance of uncooked food, especially fish, and care regarding contact with domestic animals should be enjoined.

B. INTESTINAL TENIASIS

There are numerous cestodes inhabiting the intestines of man which, while often reported from tropical and subtropical countries, have no claim to be regarded as in any way peculiar to warm climates. Several species, however, may be regarded as more or less directly tropical parasites. These will be taken up in order.

(1) *Diplogonoporus grandis.*—This Dibothriocephalid parasite has been twice found in Japan. The genital organs are disposed in double series, that is, two sets in each segment. The segments themselves are very short and broad. The genital pores open in longitudinal genital grooves. The uterus has only two prolongations to each side.

(2) *Tænia africana.*—The scolex is very small, quadrilateral, and unarmed, being provided with five suckers, one of which is apical. The segments are broader than they are long. The genital pores are irregularly alternate, being placed at the borders of the segments. The uterus has from fifteen to twenty regular prolongations. The eggs are spherical. It has been reported in German East Africa. I have a headless specimen of what is probably the same species from Rhodesia.

(3) *Tænia philippina.*—This worm measures from 80 to 100 cm. in length by one cm. maximum width. The head is cubical and unarmed. It has four suckers. The parasite was found by Hare in a prisoner in Bilibid prison in the Philippines.

(4) *Tænia bremneri.*—The segments measure about 28.6 by 8.5 mm. on an average. The head is not known. The worm was found in a Fullain woman in Northern Nigeria.

(5) *Tænia hominis.*—The scolex measures 1.34 mm. in length by 2 mm. in breadth. The rostellum is unarmed. The edge of the suckers is marked by a characteristic surrounding ridge. The entire length is stated to be 70 mm. Form of the genital organs not known. Reported from north of Persia.

(6) *Hymenolepis nana.*—The head is minute, the rostellum retractile, and the genital pores single, marginal, and unilateral. The species is very small, 5-45 mm. by 5-1 mm., the segments are broader than long. The uterus nearly fills the gravid segments. The scolex is armed with twenty to thirty hooks. It has a pretty wide distribution throughout the tropical and subtropical regions of both hemispheres, and is not uncommon in the southern portion of the United States.

(7) *Hymenolepis diminuta.*—The head is small, club-shaped, and has a rudimentary rostellum with four suckers, and is unarmed. The worm measures from 20 to 60 cm. in length and about 3.5 mm. in width at its widest point. The eggs are oval and measure about 60 μ in diameter. It is reported from South America, as well as various temperate countries.

(8) *Davainea madagascariensis.*—The rostellum is armed with two rows of hammer-shaped hooks, and the suckers are also armed with several rows of hooklets. The head is retractile, and the number of hooks about ninety. The worm is about 25-30 cm. by 1.5 mm., and the segments number from five to six hundred and contain calcareous corpuscles. The genital pores are unilateral. This species is reported from widely separated regions of the old and the new world tropics.

(9) *Davainea asiatica.*—This species measures about 30 cm. in length and is composed of about seven hundred and fifty segments. The segments measure 0.16 mm. by 1.78 mm. The posterior margin of each segment overlaps the anterior margin of the following segments. The genital pores are unilateral. The uterus is broken up into sixty to seventy egg masses in each segment. Reported from north of Persia.

D. grandis is quite possibly due to eating uncooked fish. Infection with *T. africana* is thought to arise from eating the raw flesh of the zebu. The carrier of *T. hominis* is not known. *H. nana* is found in brown and black rats, mice, field mice, and dormice, as well as in man. White rats may be easily infected. The worm may also be directly conveyed from person to person, and autoreinfection may also take place. Blanchard has suggested that cockroaches may be responsible for the spread of *D. madagascariensis.*

Little is known of the pathogenesis of several of the foregoing parasites. *H. nana* invades the villi of the intestines and is said to injure the follicles of Lieberkühn. According to Grassi, it may bore deeply into the intestinal mucosa. As the worms are very numerous the effects may be severe. Abdominal pain, occasional diarrhea, epileptiform convulsions, headache, and various nervous symptoms are described. At autopsy the mucosa of the small intestine has been found to be tumefied and hyperemic.

Remedial Measures

Male fern is almost a specific for tapeworms. Before the administration of the remedy the patient should be kept upon light diet for a couple of days, and preferably should fast for the last twenty-four hours. The bowels should be emptied by giving castor oil or Epsom salts. A good routine is: empty intestinal canal, castor oil the night before, no breakfast, anthelmintic on empty stomach.

The male fern may be given in capsules of the oleoresin, one fifteen-grain (1.0-gm.) capsule being administered every ten minutes until half a dozen have been taken. Others give a single dram (4.0 gm.) dose. In women and children and in the case of men who are weakened from disease much smaller amounts should be used. Riesman warns against using castor oil for expelling the worm on account of the solubility of male fern in oils. He suggests calomel or magnesium citrate.

After treatment has been given, and the patient allowed to rest for an hour or so, another dose of salts should be administered and the resulting stool examined for parasites.

The parasites should be passed into a vessel containing warm water, otherwise the nervous organization of the tapeworm may result in its breaking in two and the head being left unpassed. To avoid this some recommend placing gauze on top of the water in the vessel to catch the worm and avoid traction. Under no circumstances should the worm be pulled from the rectum. If it breaks and the head is left in the bowel a salt enema will sometimes result in its expulsion. In case the head is not obtained the treatment should be repeated.

Extreme weakness, acute inflammation of the gastrointestinal tract, grave cardiac or renal disease, and pregnancy are contraindications to the use of male fern. Overdoses of the drug, even in healthy persons, may set up vomiting, diarrhea, collapse, blindness, jaundice, convulsions, etc.

As substitutes pumpkin seed, Kamala, oil of Filmaron, kousso, pomegranate root, or its alkaloid, pelletierin (tannate), may be given instead.

Benzene is also recommended. Of these remedies pumpkin seed is the only one which can be safely given in pregnancy.

If necessary, tonics and supportive treatment should follow the expulsion of the worm, with iron and arsenic if anemia is present.

Preventive Measures

Personal cleanliness, the careful cooking of fish and meat, care in the selection of drinking water, and avoidance of contact of natives or animals are all to be recommended. Houses should be kept clear of mice, rats, and other vermin, and such should be destroyed. In addition to infesting rats and mice in its adult stage, *H. diminuta* is said to live in its cysticercus stage in various insects (moths, earwigs, and beetles), and such insects should accordingly be destroyed in houses. One should avoid sleeping in the same bed with a person infected with *H. nana,* and in endemic districts great care should be taken to keep all food out of the reach of mice and rats. Reisman makes the excellent suggestion that something regarding the methods of tapeworm infection and avoidance be put in school textbooks of physiology and hygiene.

2. DISTOMIASIS

CREIGHTON WELLMAN

The number of flukes that occur in man in tropical countries is probably much larger than is at present known or suspected. In many warm countries it is difficult or impossible to secure necropsies, and many cases while alive do not come to European physicians. A number of species have, however, been reported from various parts of the tropical world.

A. HEPATIC DISTOMIASIS

Several tropical flukes are found in the liver and bile ducts of man. The most important of these are the three following:

(1) *Opisthorchis noverca.*—This fluke, which is on the average 11 mm. by 2.5 mm. in size, seems to be rare in man, only having been found two or three times in India.

(2) *Clonorchis sinensis.*—This fluke, which averages about thirteen millimeters long by 3.3 broad, is found throughout the Orient, in India, Japan, Korea, Formosa, China, Tonkin, Mauritius, etc.

(3) *Clonorchis endemicus* measures 6-13 mm. in length by 1.8-2 mm. in width. The eggs measure on the average about 26 μ by 15 μ and have a flattened operculum.

Opisthorchis sinensis inhabits the gall and bile ducts and is sometimes found in the pancreas and intestines. Occasionally the infection is very heavy, as high as 4,000 flukes having been found in one person. The presence of the flukes results in inflammation, dilatation, and sacculation of the bile ducts, and interstitial hepatitis, which is accompanied by atrophy and fatty degeneration of the liver cells. The functions of the liver are perverted by the pathological changes, and after a period of enlargement the organ becomes atrophic. The symptoms set up in extensive infection are chronic diarrhea, intermittent jaundice, and later cachexia accompanied by general edema. While the course of the disease is prolonged, yet it may prove fatal.

In cases of *O. noverca* the parasites appear in large numbers in the bile ducts, which are in consequence thickened and sacculated. *C. endemicus* also occurs in cats, dogs, pigs, as well as in man. The symptoms set up are diarrhea and enlargement of the liver.

Remedial Measures

Removal of the patient from the endemic district or the institution of rigorous measures to prevent reinfection are logical procedures. Different drugs have been tried without success. In view of the beneficial effects reported from the use of salol in hepatic distomiasis of sheep, this remedy has been suggested in the present disease. Probably it should be tried in case the parasites are recognized by clinical signs or by ova in the feces. The various so-called cholagogues have also been recommended. Forchheimer suggests a choleocystotomy by means of which local treatment may be carried out.

Preventive Measures

The mode of infection is unknown, and no specific measures can be advised. Cleanliness, the cooking of food, and the boiling of water are, of course, wise on general principles. *Opisthorchis noverca* is said to be found in the liver of dogs in India. *C. endemicus* infests cats and pigs besides dogs.

B. INTESTINAL DISTOMIASIS

A number of tropical trematodes are parasitic in the intestines of man. The most important of these are as follows:

(1) *Gastrodiscus hominis.*—This parasite is a small fluke averaging about 6.5 mm. in length and 3.5 in breadth. The anterior portion is subcylindrical or roughly conical, while the posterior portion presents the appearance of a disk flattened and concave on its ventral surface. It is

probably not a regular parasite of man, but is doubtless normally dependent upon some common mammal. It has been observed, however, in an Assamese from Gowhatly and an Indian in Tirhoot.

(2) *Clonorchis watsoni.*—This parasite infests man in West Africa and is about 9 mm. by 4.5 in size. Manson believes it is not uncommon, especially in children. Probably similar African flukes, e. g., the common Angolan antelope fluke described by Stiles and Goldberger under the name *Wellmanius wellmani,* will occasionally be reported in man.

(3) *Fasciolopsis buski.*—This, the largest known trematode infesting man, averages about 30 mm. in length by 11 mm. broad. It has been reported from India, Siam, Sumatra, China (Canton), and Hong Kong.[1]

(4) *Fasciolopsis rathouisi.*—This species is similar to *F. buski,* but averages only 17 mm. in length by 9.5 mm. broad. It occurs in China (where it is called Siau San Dzoong), and other parts of the far East.

(5) *Fasciolopsis füllerborni* measures from 30 to 50 mm. long by 14 to 16 mm. broad. The eggs average 1 mm. in length by 0.073 mm. in width. This large parasite was found in the feces of a Hindoo in Hamburg.

(6) *Fascioletta ilocana* measures from 4 to 6 mm. in length by 0.75 to 1.35 mm. in width. The eggs measure from 88.8 μ to 114.7 μ in length by 53.5 μ to 81.9 μ in breadth. This small species was discovered by Garrison in the feces of man in Luzon, Philippine Islands, in 1907.

(7) *Heterophyes heterophyes.*—This small parasite, which measures only about 1.5 by 5 mm., is probably of wide distribution, having been reported from such widely separated countries as Egypt and Japan.

Gastrodiscus hominis was found to be present in the cecum and colon in large numbers, the mucous membrane showing small red spots where the flukes had been attached by means of their suckers. *Clonorchis watsoni* was found by Watson, on *post mortem,* in the small intestine, the jejunum being full of flukes, some of them still adherent to the mucosa, which showed no distinct hemorrhages, but seemed somewhat congested. Before death the patient had numerous watery stools, deeply colored with bile and containing many adult flukes. *Fasciolopsis buski* is parasitic in the duodenum of man, and its presence is said to cause fever (with temperature rising as high as 106° F.), bloody diarrhea, emaciation, lymphadenitis, edema, and other typhoid symptoms. It is in many instances fatal. The presence of *F. rathouisi* is said to cause diarrhea, light-colored stools of very offensive odor, tympanitis, wasting, and occasionally jaundice. Among the Chinese infection with this fluke is thought usually to cause death. *Heterophyes heterophyes* inhabits the small intestine, usually the central third of the jejunum, but does not seem to cause serious symptoms.

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[1] Apparently very similar to *F. buski* is an unnamed trematode described by Heanley. This fluke was found by a Chinese physician in a child in Hong Kong and is two inches in length by one-half inch in breadth. The cuticle is armed with spines and the genital organs closely resemble those of *F. buski.* The flukes were vomited by the patient.

Remedial Measures

In case the presence of intestinal flukes is suspected or indicated by the patient's passing adult worms or eggs, some anthelmintic, such as thymol, male fern, eucalyptus oil, or, as some recommend, chloroform, may be given while the patient is fasting. Such vermifuge should be preceded by a laxative and followed by a purge. Such treatment is specially successful in the case of *Fasciolopsis* infection, and the worms are expelled in large numbers. The purge employed may be Epsom salts, although when eucalyptus oil is employed the purgative had better be castor oil.

Preventive Measures

The life cycles of *G. hominis* and *C. watsoni* are unknown, but probably uncooked food or infected water should be considered as dangerous. In the Orient animals should not be allowed near or about dwellings. The life history of other Paramphistomidæ suggests that avoidance of raw fruit and vegetables, especially water cress, and boiling water before drinking are measures to be adopted in endemic regions. *F. buski* is said to be very common in South China in pigs. Living in proximity to these animals should be avoided and cooking food and boiling water, therefore, naturally occur to one as sensible prophylactic measures. Goddard believes that uncooked vegetables or raw fruit are involved in the spread of *F. rathouisi.* It is probably common to man and some domestic animal. Cleanliness, cooked food, boiled water, and keeping animals at a distance would seem wise measures against the spread of this species. As *H. heterophyes* seems to be common to several animals besides man (having been reported in dogs, foxes, kites, etc.), a rational prophylaxis would consist in the avoidance of these animals, and in cleanliness, especially the boiling of drinking water and cooking of food.

3. ENDEMIC HEMOPTYSIS

Creighton Wellman

Paragonimus westermanni measures about 7 mm.-17 mm. by 4 mm.-9 mm. The eggs are operculated and measure about 0.9 mm. by .06 mm. The conditions arising from its presence in the lungs are often seen in the far East, and two cases from the Pacific coast region, one reported by myself, have been observed. My own case came from Formosa, where the disease is endemic, a considerable part of the population being infected. It is probable that the disease will gain a foothold in the United States.

The infected patient usually has a chronic cough which is occasionally accompanied by attacks of hemoptysis and often a considerable degree of anemia. The hemoptysis may be slight or considerable. The lungs are tunneled by the parasites and infiltrated by their ova, and there is no hope of curing the condition. Mild cases, if reinfection is avoided, may recover spontaneously. Palliative and symptomatic treatment with general tonic measures are, therefore, all that can be aimed at.

PREVENTIVE MEASURES

Sputum and other discharges containing ova should be burned. In endemic areas water should be boiled before drinking, and no uncooked food should be eaten. As domestic animals, such as cats, dogs, pigs, etc., harbor the parasite, both in the Orient and here in the United States as well, association with these should be avoided.

4. SCHISTOSOMIASIS

CREIGHTON WELLMAN

There are three species of *Schistosomum* known to affect man in tropical and subtropical regions. The diseases arising from the presence in the tissues of these different worms will be considered separately.

A. ENDEMIC HEMATURIA

This disease is due to *Schistosomum hæmatobium* and is found in Africa, Mauritius, and the near East. *S. hæmatobium* is flattened in contour, and the male measures 11 mm.-15 mm. by 0.9 mm.-1.1 mm. The female is rounded and measures about 18 mm.-20 mm. by 0.2 mm.-0.3 mm. The eggs are oval and have a terminal spine. They measure about 0.12 mm.-0.2 mm. by 0.05 mm.-0.07 mm. The integument of the male is tuberculated. The parasites are found in the portal vein and its branches, the veins of the bladder, and of other pelvic organs. The female deposits her eggs in the smaller veins, especially in the walls of the bladder, and these eggs escape through the mucous membrane into the lumen of the bladder and are passed with the urine. Other organs, notably the liver and rectum, are also invaded.

The conditions set up by the presence of the parasite are papillomatous-like growths in the mucous membrane of the bladder, ulcers, thickening of the bladder walls, etc. Some blood is usually passed with the urine, generally to be seen in only the last few drops passed. Sometimes, however, the hemorrhage is extensive and the entire contents of the bladder may be colored with blood. Urinary fistulæ, calculi, urethral stricture, etc., may be produced. In many cases, however, the presence of the parasite gives rise to little discomfort, and most of the long series of cases seen by me in West Africa were of a mild type.

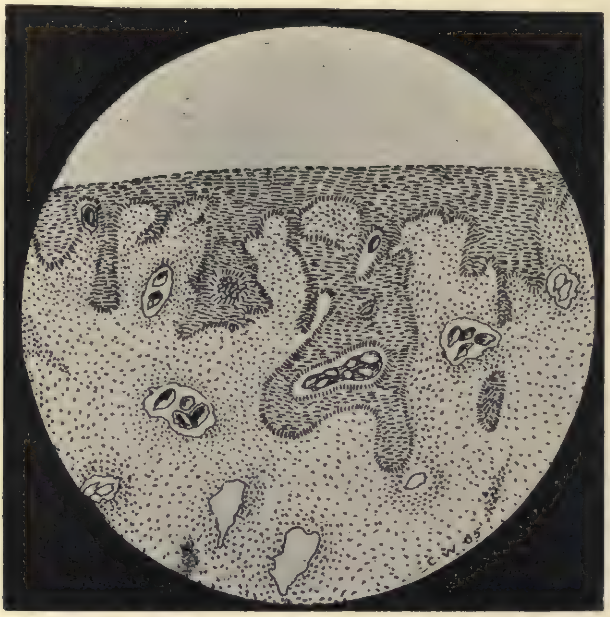

Fig. 1.—Schistosomal papilloma of the Rectum. Section showing ova of *S. hæmatobium in situ.* (After Wellman.)

Remedial Measures

No specific treatment can be attempted. Some authors have advocated the use of male fern, methylene blue, etc., but results do not appear to be encouraging. As Manson observes, the treatment resolves itself into that of chronic cystitis. Urotropin in doses of five grains (0.30 gm.) three times a day may be tried.

In a long series of cases treated by me in West Africa I found excellent results from the exhibition of hyoscyamus, probably on account of its sedative effect in the prevention of vesical tenesmus. Where the hematuria is excessive, adrenalin may be tried to control it and sedatives may be indicated for the pain which is sometimes present.

Irrigation of the bladder with a boric acid solution (1 per cent.), followed by 1:5,000 solution of nitrate of silver, has been very beneficial in my experience.

Calculi, fistulæ, stricture, etc., should be dealt with surgically. In severe cases rest in bed and tonics are indicated. The patient should be encouraged to take much liquid. Nourishing, non-irritating food should be recommended, stimulants and spices being avoided. Overexertion and other excesses, chill, and all physiological depression should be guarded against. I have found that the administration of cod liver oil is usually of great benefit in this chronic and exhaustive disease.

Preventive Measures

In endemic areas one should avoid bathing in streams, pools, and public baths. The urine and feces of patients should be destroyed or disinfected. Natives should be specially instructed against urinating into streams or other collections of water. All food and drinking water should be cooked or filtered, and the source of supply furnishing water for bathing and other domestic purposes should be carefully inspected.

B. Intestinal Schistosomiasis

This disease occurs in Africa and tropical America, including the West Indies, and is due to infection with *Schistosomum mansoni.* The male measures about 12 mm. by 0.44 mm., and the female about 14.5 mm. by 0.16 mm. The eggs measure 146 μ by 62 μ and have a lateral spine.

This parasite is found principally in the mesenteric veins. The eggs being deposited in the submucosa of the intestines, especially the rectum, give rise to infiltration and papillomatoid growths, which are accompanied by various symptoms. Diarrhea and dysentery are most frequently seen. Occasionally other organs, notably the lungs and the liver, may also be affected. In case of such latter involvement a peculiar fibroid pneumonia and cirrhosis of the liver are found.

Remedial Measures

As in the case of endemic hematuria, no specific treatment can be attempted, and palliative measures are the most that can be hoped for.

In case infiltrated tumors of the rectum and other intestines can be felt, these should be treated surgically. Enemata of quinin and sodium hypochlorid (1:100) have been recommended when there are ulcers low down in the bowels.

Preventive Measures

These are the same as those for the prevention of endemic hematuria.

C. KATAYAMA DISEASE

This affection is caused by a third species, which probably has a wide distribution in the Orient (*Schistosomum japonicum*). The male measures 9 mm.-12 mm. by 0.5 mm., and the female 12 mm. by 0.4 mm. The eggs measure 70 μ-75 μ by 45 μ-55 μ, and are smooth and without any prominent spine. The integument of the male is not tuberculated.

The adult parasites seem to inhabit the smaller blood vessels of the mesentery, and the ova are found in the mucous and submucous coats of the intestine principally. There may be, however, involvement of the liver, mesenteric glands, pancreas, and almost every other organ of the body, including the brain. The bladder has not been shown to be affected. When the brain is involved, headache, vertigo, nervous manifestations, Jacksonian epilepsy, and paralysis may all appear.

Remedial Measures

As in the case preceding, no specific treatment is available.

Preventive Measures

Since it has been shown that the cat is a carrier of *S. japonicum,* destruction of these animals in endemic districts should be insisted on. Avoidance of uncooked food and impure water is also to be considered.

5. FILARIASIS

WILLIAM B. WHERRY

The failing interest of the medical profession, in the rôle played by animals in the production of disease, was most successfully revived when the white man invaded the tropics. One by one the causes or means of transmission of many hitherto obscure diseases were found to fall into this group. The precise information concerning the life history of these parasites, the acquisition of which necessarily precedes rational prophylaxis, was largely the result of the investigations of zoölogists. But in many instances the pages record the labors of the physician in the far parts of the earth.

FILARIA BANCROFTI

Prophylaxis

Through the brilliant experiments of Manson in China, Bancroft in Australia, Low in the West Indies, and James in India, it has been definitely established that certain mosquitoes act as intermediate hosts for the embryos of *F. bancrofti.* The embryos *F. nocturna,* circulating with the peripheral blood of the human host, are taken up by various species of *Culex* and *Anopheles.* In the tissues of these insects they develop and undergo a metamorphosis, which necessarily precedes their reintroduction into a human host. The most widely distributed of these mosquitoes is *Culex fatigans, var. skusii,* a voracious night feeder, which acts as intermediate host in Asia, Australia, the West Indies, and elsewhere. James showed that *Anopheles rossi, Culex microanulatus,* and *Culex albopictus* were favorable hosts in India. According to Annett, Dutton, and Elliott, *F. nocturna* undergoes complete metamorphosis in *Anopheles costalis* in West Africa. The theory that infected mosquitoes reinoculate the human host during a subsequent feeding received support from the domain of comparative pathology. Grassi and Noè were able to produce infection in muzzled dogs which had been subjected to the bites of mosquitoes infected with the larvæ of *Filaria immitis* (see also Fülleborn, 1908a). Hence, prophylaxis here coincides with that against malaria. Protecting the mosquitoes against infection, by screening human hosts, is almost as important as culicidal measures.

Treatment

The treatment of the various lesions resulting from the obstruction of the lymphatics has perhaps some hope of now being transferred from the surgical side to the medical. Attempts to kill the parasites with thymol, extractum felicis, methylene blue, benzoic and boric acids, sodium benzoate, picric-nitrate of potash, etc., have been abandoned as failures. The usual termination of hematochyluria was suicide; of varicose glands, recurrent lymphangitis, often followed by elephantiasis.

McDill and the writer treated a case of hematochyluria with the X-rays and, at the suggestion of Musgrave, cinchonized her before applying the rays. This patient recovered. The embryos disappeared from her blood, and she was in perfect health four years later.

Wellman and Von Adelung tried this method with apparent success, but the latter reported on its failure later on.

It appears, however, that recent arsenical preparations kill the adult worms. This means much, for the early destruction of the parasites promises relief from many hopeless conditions and from the surgical intervention which is so often the last resort. O'Brien killed off the embryos *F. nocturna* from the circulation of a Cingaleese with atoxyl; and, as they did not reappear during a year's observation, it is presumable that the parent worms were killed. This case received intravenous injections of 5 minims, gradually increased to 40 minims, of a 5 per cent. solution in sterile saline. It was given at first every three or four days, and later every day, the whole treatment lasting about six weeks.

Reichmann gave 0.3 gm. "Ehrlich-Hata" to a patient suffering from pleurisy with an undetermined filaria-like worm in the pleural exudate. This was followed by recovery.

Pilcher and Pilcher treated a white female, 35 years old, who developed chyluria four years after leaving the tropics. Chylous urine was secreted from the left ureter only. She was given 0.6 gm. "606" hypodermatically without any marked constitutional derangement. The filarial embryos, which appeared only at night in the peripheral circulation, and hence were probably the embryos of *F. bancrofti,* disappeared within twenty-four hours after the injection. Between the sixty-eighth and seventy-second hours following the treatment the urine became markedly chylous and contained a large number of dead embryos and thread-like adult parasites.

Werner (1911) reports a failure after the intravenous injection of 0.5 gm. salvarsan. The *F. nocturna* were not diminished in numbers three days after treatment.

For surgical technique in connection with filariasis, the reader is referred to the articles of Charles, Werner, Stokes, and Lanz.

DRACUNCULUS MEDINENSIS (GUINEA WORM, MEDINA WORM, THE WORM OF PHARAOH, ETC.)

Prophylaxis

The idea that infection with the guinea worm is acquired by drinking polluted waters can be traced back to antiquity. O'Donovan, who explored the Merv oasis, records the statement of a Khan of the Turcomans, "that anyone who drinks the stagnant water of the rain pools, or that of the tanks of the caravanserais (in southern Bokhara), is certain to take into his system the egg of the *rishté,* which will then infallibly develop itself." Several observations point to the incubation period in man being from eight months to a year.

The experiments of Fedschenko in Turkestan, which have been confirmed by Manson and Blanchard, point to certain fresh water Cyclops (*Cyclops quadricornis, strenuus, viridis, bicuspidatus*) as the intermediate host. The filarial embryos escaping into water pass through the integument and into the body cavity of the Cyclops. There they undergo a metamorphosis somewhat similar to that of *F. nocturna* in the mosquito. Wenyon confirmed the observation of Leiper, that the addition of very dilute hydrochloric acid caused the completely metamorphosed embryo to desert the Cyclops—thus simulating the probable course of events in the human stomach. These experiments make it almost certain that infection is acquired through drinking water. However, as Plehn was apparently successful in infecting a monkey by feeding the undeveloped larvæ, one should guard against more direct methods of infection, as well as adhering to the use of boiled or carefully filtered water.

Treatment

The ancient method of gradually extracting the untreated worm by winding it on a split stick should be avoided. Accidental rupture of the parasite is followed by serious results—intense inflammation and suppuration, often followed by bone necrosis, septicemia, or contractions and ankyloses.

In the endemic regions natives who make a vocation of extracting the worm acquire great skill. With their small light hands they knead the worm toward the opening and seldom break it. According to Maclean, the practice of douching the part with cold water to aid extraction was universal in the endemic regions in India. There is no doubt that this practice hastens the emptying of the worm's uterus, and so minimizes the dangers incident to extraction. Manson recommends the following procedure: the site occupied by the parasite should be douched with cold water three or four times a day, and in the meantime protected by dressings wet with water. As a result of this stimulation the uterus empties itself more rapidly. Complete evacuation is assured when the worm no longer reacts to the water test. Now extraction is comparatively easy.

However, the method of Emily is perhaps the best and safest, and its value has been amply confirmed. If the worm is protruding, a one to one thousand solution of bichlorid of mercury is injected, by means of a fine syringe, into its body; twenty-four hours later it may be extracted with ease. If the worm is not visible, a few drops of the same solution are injected as near it as possible. The worm dies and is absorbed, or may then be extracted with the aid of a knife.

FILARIA LOA

It is not known just how infection with *F. loa* takes place. The weight of evidence points to *F. diurna* as the embryo of *F. loa*. This is supported by Huffman, who studied the embryos extracted from a live loa.

On the assumption that the embryos of *F. loa* circulate in the daytime, Manson has suggested that some day-biting insect, such as the Mangrove fly (*Chrysops dimidiatus*), must act as intermediate host.

It seems certain that *F. diurna* must be a different embryo from *F. nocturna,* for Annett, Dutton, and Elliott found that the latter developed in *Anopheles costalis,* while the former did not.

Brumpt did not succeed in developing *F. diurna* in a species of *Glossina,* but the list of biting flies in the endemic region has by no means been exhausted. That the disease is perhaps only acquired in "the bush" may follow from information acquired by the writer from Dr. Emil Blunden, long a missionary at Batanga, German Cameroon, West Africa. He felt that there was much *a priori* evidence that loa infection followed the bites of certain flies, and cited as evidence that, according to his observations, when women accompanied their husbands on itinerary expeditions they were severely bitten by flies, and that all these women who travel about much become infected with loa. Their husbands, who lead a more active life, are less severely bitten, and often escape infection. Further, the wives of government officials who stay at home escape infection.

Treatment

Usually the presence of the loa is not suspected until it is seen making its way beneath the skin of the body or extremities, or under the conjunctiva. Its passage beneath the conjunctiva is usually accompanied by a stinging pain and slight edema. Persons harboring the loa usually complain of the repeated appearance of single or multiple swellings on various parts of the body—especially the extremities. These are the so-called "Calabar swellings." Manson has brought forward evidence to show that these are due to the subcutaneous discharge of the embryos of the loa—the resulting edema being analogous to that following the rupture of the Medina worm. In the case of the *loa,* however, the swelling is transitory. Taken altogether, the symptoms hardly call for medical treatment. However, it is customary, particularly in the case of neurasthenic whites, to remove the parasites one after another, as opportunity offers. When one appears beneath the skin or conjunctiva, it is seized and held by forceps while cocain is applied. Then it is cut down upon and removed. This should be done without rupturing the parasites, for rupture is followed by intense local edema which, in the case of the eye, is uncomfortable, and, though not dangerous, requires treatment for three or four days.

Perhaps rupture of the male is not followed by edema. Dr. Blunden ruptured a loa while removing it from the conjunctiva, and there followed an intense edema. This worm was one of the female loas described by Huffman and the writer.

FILARIA PERSTANS

Wellman found what he believed to be developmental forms of this filaria in ticks (*Ornithodorus moubata*) fed on infected blood. His experiments were performed with ticks bred from eggs and conducted more carefully than those of Feldman. Fülleborn (1908b) was unable to confirm these observations, and also had failures with *Stegomyia fasciata, Culex pipiens,* and *Pulex irritans.* He refers to the work of Noè, who had reported the metamorphosis of the Filaria Grassi of dogs in a tick.

6. OTHER NEMATODE INFECTIONS

CREIGHTON WELLMAN

The cosmopolitan roundworms, such as *Ascaris lumbricoides, Oxyuris vermicularis, Trichinella spiralis,* etc., may be found, often in enormous numbers, throughout the tropics. Still, such species cannot be considered as in any way special to hot climates. The following forms, however, merit special attention in a work devoted to the treatment of tropical diseases.

A. HOOKWORM DISEASE

This infection is a frequent cause of anemia and death in the tropics. In one district in tropical Africa investigated by me thirteen per cent. of the population were infected. In addition to the old world hookworm (*Anchylostoma duodenale*) I found in the same district the new world form (*Necator americanus*) as well. This last form seems to be almost cosmopolitan, as it was sent to me from East Africa and Rhodesia and has been reported from South America, Australia, the Philippines, and other parts of the far East, as well as from the United States and Europe.

The human species of hookworms are very similar in general appearance and are elongated cylindrical worms, the mouth being provided with six papillæ and the esophagus not forming a distinct bulb. The males are provided with a caudal bursa. The two forms may be briefly diagnosed as follows:

(1) *Anchylostoma duodenale.*—This worm measures about 8 mm.-11 mm. by 0.4 mm.-0.5 mm. (male), and the female 10 mm.-13 mm. by 1 mm. The eggs measure 64 μ-72 μ by 36 μ-46 μ. The buccal capsule is provided with four ventral hooks and two dorsal teeth. The tail of the female is armed with a delicate spine. That of the male with a trilobate bursa having eleven ribs and two spicules. The bursa has a dorsomedian

lobe and prominent lateral lobes united by a ventral lobe. The dorsal ray divides at a point about two-thirds its length from the base, and each branch is tridigitate. This is the old world hookworm. A number of species similar to this and the following occur in animals.

(2) *Necator americanus.*—The males measure about 7 mm.-9 mm. by 0.3 mm.-0.35 mm., and the females 9 mm.-11 mm. by 0.4 mm.-0.5 mm. The eggs measure 52 μ-61 μ by 32 μ-38 μ. The dorsal lobe of the male caudal bursa is short and divided at the tip. The dorsal ray is divided to the base and the tips are bipartite. There is one pair of semilunar ventral buccal plates, smaller than in *A. duodenale.* A dorsal conical median tooth projects into the buccal cavity. There are also one pair of dorsal and one pair of ventral submedian lancets deep in the buccal capsule. This is the new world hookworm.

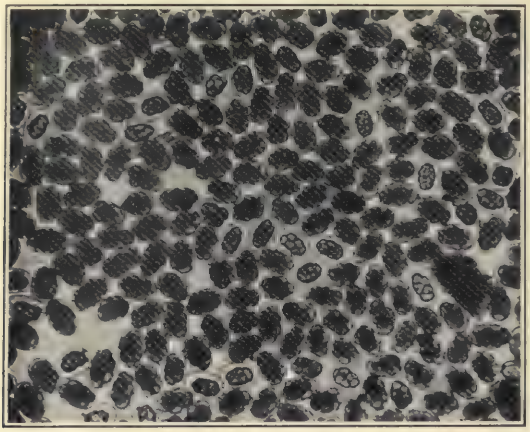

Fig. 2.—Hookworm Eggs Obtained by Centrifugalization of Feces. (After Bass.)

The worms inhabit the small intestine. They are found most abundantly in the jejunum, but they also occur in the duodenum and ileum. They attach themselves firmly to the mucous membrane of the intestine, but are sometimes found free in the canal. The eggs are excreted with the feces and may retain their vitality for long periods, especially under conditions of moisture, warmth, and darkness. Sunlight and desiccation destroy them. On one occasion I observed the larvæ free and escaping from eggs in recently passed feces, which is, so far as I know, a novelty.

The most usual mode of infection is through what is known as ground itch. In the case of barefooted natives in regions where the disease is endemic the larvæ which have been hatched out in mud or slush from polluted soil penetrate the skin of the feet and legs and produce a dermatitis which afterward becomes vesicular and later pustular. The larvæ giving rise to this condition penetrate the skin and subcutaneous tissues, and enter the blood vessels, from whence they are carried to the lungs. From here they pass by way of the bronchioles, bronchi, and trachea to the larynx. From the larynx they pass into the throat, down the esophagus, through the stomach, and into the small intestines, the site of their normal habitat.

In West Africa ground itch is not uncommon in the districts where the hookworm occurs. The native black is most often affected, but suffers less from the effects of the disease than does the European. The conditions set up by the presence of the parasite are often serious. Pain and uneasiness in the abdomen, usually with some tenderness, headache, and dizziness, and a capricious appetite, are usually complained of. There may be nausea, vomiting, and some fever. The patient tires easily and there is anemia and later edema. Heart weakness often is marked. The patient may remain plump, but muscular weakness is very pronounced. Infantilism and retarded mental development are common results of infection in the young. In fatal cases fatty degeneration of the parenchymatous organs is found at autopsy.

Remedial Measures

As Dock and Bass in their excellent monograph point out, "It is important to understand the conditions that must be met in the treatment of hookworm infection." We must remember that the worms are in the small intestine, their heads imbedded in the mucosa, and their bodies surrounded by mucus and food. Therefore, the gut must be cleansed and emptied of food and mucus and the anthelmintic brought into direct contact with the worms before we can hope to expel them.

A number of drugs have been recommended for the expulsion of the parasite. Among these are thymol, oil of eucalyptus, betanaphthol, turpentine, etc. Thymol has been found to be the most effective and practical of these remedies, and should always be chosen unless the patient has some idiosyncrasy which prevents him from taking it. Before taking the drug the patient should fast during the whole or part of the preceding day, and the night preceding the administration of the thymol he should have a generous dose of Epsom or other salts.

The thymol should be administered upon an empty stomach and should consist of forty to sixty grains (3.0 to 4.0 gm.) divided into three portions, taken about an hour apart. An hour after the last portion has been taken a second dose of Epsom (or other) salts should be given. Not less than 60 grains (4.0 gm.) should be given an adult, unless great weakness is present, and children's doses should be in proportion.

The following table of doses from Ashford and King is recommended by Stiles, and also Dock and Bass, as being a safe and practical guide:

Ages	Grains	
Under 5 years old (in size)	7½	(0.5)
5 to 10 " " " "	15	(1.0)
10 to 15 " " " "	30	(2.0)
15 to 20 " " " "	45	(3.0)
20 to 60 " " " "	60	(4.0)
Over 60 " "	30-45	(2.0 to 3.0)

It is well to triturate the thymol thoroughly with sugar of milk before administering it. The thymol being soluble in fats, alcohol, ether, glycerin, vinegar, chloroform, etc., these substances should not be ingested during the treatment lest the patient show symptoms of thymol poisoning.

As regards time of administration, Stiles and Dock and Bass all advise choosing Sunday as being convenient to working people, school children, etc. Dock and Bass recommend the following instructions to be given the patient:

All of Saturday, milk or other light diet.
Saturday night, 1 ounce (32.0 gm.) sodium sulphate.
Sunday, 6 A. M., 20 (1.3 gm.) grains thymol.
Sunday, 7 A. M., 20 (1.3 gm.) grains thymol.
Sunday, 8 A. M., 20 (1.3 gm.) grains thymol.
Sunday, 10 A. M., 1 ounce (32.0 gm.) sodium sulphate.
Drink nothing but a little water until 4 P. M. and remain in bed.
After 4 P. M. rise and eat what you want and do what you like.

It is usually best to repeat the treatment in about a week or ten days. Three or even more treatments may be necessary.

The symptoms of thymol poisoning which may supervene if a solvent of the drug be accidentally taken are lassitude, headache, vertigo, ringing in the ears, nausea, cardiac weakness, etc.

For the anemia and debility remaining subsequent to the specific treatment, iron should be administered. Blaud's pill or the tincture of iron has been found in my experience to be the most effective. The latter may be pleasantly taken in lemonade. In the case of children syrup of the iodid of iron may give more satisfactory results than the preparations just mentioned.

In case oil of eucalyptus, turpentine, male fern, podophyllin, kerosene, or betanaphthol are used to expel the worms, the same elementary treatment should be employed as when thymol is used.

Preventive Measures

In the words of Dock and Bass, the problem of hookworm prophylaxis involves the following essential elements:

"1. Stopping the danger of infection by exterminating the mature worms in the bodies of human beings in order to check the supply of eggs at the source.

"2. Preventing the growth and existence of larvæ in the places where they develop.

"3. Preventing infection by larvæ that have developed." For larvæ will develop in spite of all efforts that can be put forth at this stage of our sanitary knowledge and influence.

The great mistake often made is to treat only those with severe evidences of the disease and to allow "hookworm carriers" to go about undiagnosed and untreated in endemic areas. All affected persons, whether sick or apparently well, should be examined and treated.

The prevention of soil pollution is of the greatest importance. Among the greatest needs of civilized and semicivilized communities as measures to prevent soil pollution is some means to provide for the proper disposal of feces. In many countries sanitary privies may be devised and property holders required to build and maintain these. The specifications worked out by the officers of the United States Public Health and Marine Hospital Service are simple, and the buildings inexpensive, and these are recommended to Boards of Health and Colonial Medical Officers.

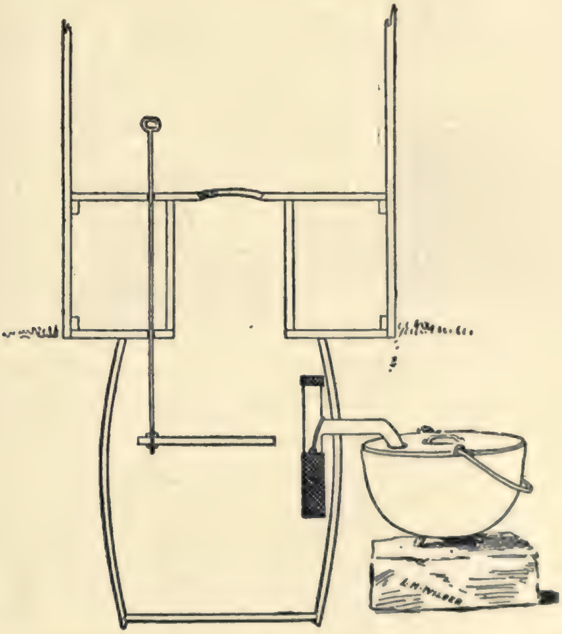

Fig. 3.—Wet System Sanitary Privy. (After Lumsden, Roberts, and Stiles.) The container is an ordinary barrel sunk in the ground. The antisplashing device is made of wood. The water in the container should be coated with a film of petroleum to prevent mosquito breeding.

Drainage is also of value as an antihookworm measure, and deep mines where drainage is impossible should be disinfected with common salt, or possibly even more cheaply by some of the sulphuretted waste products of the mine itself.

As other methods of infection besides the one above described are probably involved in the spread of hookworm, care should also be taken regarding cleanliness of the hands, food, and water. The water should be boiled or filtered, and uncooked food should be avoided. It has been shown that the feces of flies which have fed upon infected stools may contain the ova of intestinal parasites. Screening of houses, or at least of food, is, therefore, indicated. Finally, education in this as well as in all other sanitary movements is of the greatest importance. The object lesson of what has been accomplished among an ignorant and backward race in Porto Rico furnishes hope for all who are charged with hookworm extermination.

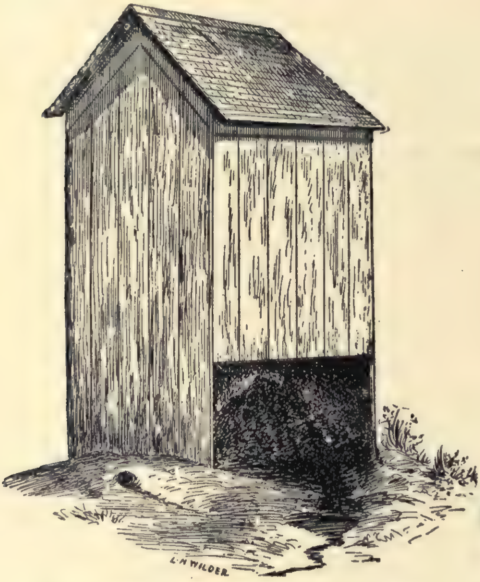

Fig. 4.—An Unsanitary Privy, Open in Back. (After Stiles.)

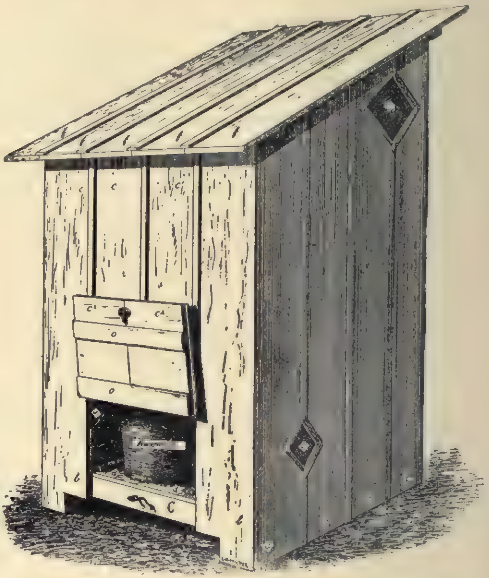

Fig. 5.—A Dry System Sanitary Privy. (After Stiles.) The door should be kept closed, the ventilators screened to keep out flies, and the seats provided with hinged lids.

B. Cochin China Diarrhea

This parasite has a geographical distribution closely coinciding with that of hookworm. The worm, *Strongyloides stercoralis,* is heterogeneous in its development and several stages, which were formerly thought to be different parasites, are found in man. The heterogamy of *Strongyloides stercoralis* has resulted in much confusion regarding its name. It is seen in two forms: (1) The parasitic generation measures about 2.2 mm. by .034 mm. The cuticle is finely striated. The mouth has four lips, the esophagus is cylindrical and about one-third the length of the body. The anus opens near the posterior extremity, the vulva at the posterior third of the body. The eggs average about .055 mm. by .032 mm. This generation is either parthenogenetic or hermaphroditic. (2) The free living generation is sexually differentiated. The body is smooth and the mouth has four lips. The esophagus is short with a double rhabditoid dilatation. The females measure about 0.7 mm. by .035 mm., the males 1 mm. by .05 mm., and the ova .07 by .045 mm.

The exact *rôle* played by the parasite in the production of disease is not well understood, but most authors agree that it gives rise to diarrhea, especially when the infection is heavy. It has also been shown recently that the worms may invade other parts of the body and cause serious symptoms and even death. The worms bore into the mucous membrane of the intestine and also into the glands of Lieberkühn. The eggs develop in the wall of the intestine and, therefore, only exceptionally appear in the feces. The mode of infection is not known, although it has been supposed that in this respect *S. stercoralis* resembles hookworm.

Remedial Measures

In view of the fact that the worms burrow into the mucous membrane of the intestine, treatment is unsatisfactory. Various anthelmintics have been used, but without much success. Small doses of thymol long continued seem to have given the best results.

Preventive Measures

Probably the prophylaxis should be the same as that against hookworm.

C. TRICHOCEPHALIASIS

Trichocephalus trichiuris, commonly known as whipworm, is very common in warm regions, in some districts over half the inhabitants being infected. The worm measures from 40 to 45 mm. (male), and from 45 to 50 mm. (female) in length. The eggs are longly oval, brownish in color, thick-shelled, with a clear plug at each end, and measure about 50 μ by 23 μ.

The worms are found in the cecum, more rarely in the vermiform appendix. The effects on the host are not thought to be serious, although many authors consider that, as the anterior extremity of the worms is often found buried in the intestinal mucosa, the presence of the worms may facilitate the entry of secondary bacterial invaders, and lead to intestinal disturbances, appendicitis, enteritis, etc. The received ideas as to the innocuousness of *T. trichiuris* would seem to need revision since the published fatal cases of Musgrave, Clegg and Polk, and of Jamieson and Lauder. The pathology of such cases appears to consist of a severe secondary anemia. Monkeys and lemurs are also hosts of this species.

Remedial Measures

Anthelmintics may be tried, although it seems to be the consensus of opinion among those who have studied the parasite that it is impossible to expel it with any degree of thoroughness. Male fern, santonin, or thymol will always rid the patient of part of the worms. Musgrave and others recommend benzene irrigations, 1-4 drams (15 to 60 c. c.) to the quart of water. Riesman considers that, "unless it can be definitely proved that the worms are causing disturbances, it is best not to persist in attemps at their removal."

Preventive Measures

Cleanliness, a pure water supply (or at least boiling or filtering of suspicious water), and the avoidance of pets which harbor the worm are all to be kept in mind.

D. STRONGYLOSIS

Several small worms belonging to the genera *Strongylus* and *Trichostrongylus* have been reported from the tropics as occurring in man.

(1) *Strongylus subtilis.*—The males of this small worm measure about 4-5 mm. by .09-.07 mm. The female measures 5.6-7 mm. by .09 mm. The eggs are oval, with thin shells, and resemble hookworm ova.

(2) *Strongylus gibsoni.*—The male measures 21 mm. in length and has the anus 0.2 mm. and the vulva 0.5 mm. from the tip of the tail. The eggs measure 110 μ by 53 μ.

(3) *Trichostrongylus instabilis.*—The male measures 4 to 5 mm. in length by about .08 mm. in breadth at its greatest diameter. The female measures 5 to 6 mm. in length by about .09 in thickness. The anus is about .07 mm. and the vulva about 1.2 mm. in front of the posterior extremity. The eggs measure 72-80 μ by 40-43 μ. The life history is unknown.

(4) *Trichostrongylus probolurus.*—The male measures 4.5-5.5 mm.

long by .08 mm. at its greatest diameter, and the female 4.5-6 mm. by 1.15, the anus being .05 mm. and the vulva 1.25 mm. in front of the posterior extremity. The eggs measure 76-80 μ by 43-46 μ. The life history is unknown.

(5) *Trichostrongylus vitrinus.*—The male measures 4 to 5.5 mm. long by .085 mm. thick at its greatest diameter. The female measures 5 to 6.5 mm. long, while the vulva is 1.15-1.25 mm. in front of the posterior extremity. The eggs measure 84-90 μ by 46-50 μ. The life history is unknown.

S. subtilis occurs in camels and men in Egypt and the Orient. *T. instabilis* and *T. probolurus* are normally parasites of sheep, antelopes, and dromedaries, the former occurring also in monkeys. Possibly the undetermined nematode embryo found by myself in the blood stream of a West African negro is to be referred to some such worm as the above. In such an event the life history probably resembles that of hookworm.

No particular ill effects have been noticed from the presence of these parasites in the intestine.

Remedial Measures

The worms are delicate and probably any vermifuge followed by a purgative would expel them.

E. Esophagostomiasis

Several species of *Œsophagostomum* have been found in man in the tropics, as follows:

(1) *Œsophagostomum brumpti.*—The female only is known and measures 8.5-10.2 mm. by 0.295-0.325 mm. Reported from West Africa.

(2) *Œsophagostomum stephanostomum* var. *thomasi.*—This worm from South America is similar in its structural characters and habits to the foregoing. It has been suggested that some of the forms of tropical anemia in Central and South America may be due to such worms as this. The species under discussion has been studied carefully and causes cystic nodules in the walls of the intestine. Romanovitch has reported septicemia in a monkey caused by esophagostomiasis.

(3) *Œsophagostomum apiostomum.*—This species resembles *Necator americanus* rather closely in size and general macroscopic appearance, but lacks the characteristic crook at the anterior end of the body, and possesses a terminal mouth which is surrounded by setæ which protrude through its aperture.

Leiper has noted extensive diarrhea and scouring in many of the lower animals caused by various species of *Œsophagostomum,* but points out that in the tropics the cysts and lesions of the epithelium associated with the presence of these worms in man might easily be overlooked. The worms encyst themselves in the walls of the cecum and colon and should be thought of when an unexplained dysentery with anemia is present.

Remedial Measures

There is no hope of expelling the two species first mentioned by means of a vermifuge. The last-named worm will probably be confused with hookworm, but if recognized can evidently be expelled with thymol.

F. OTHER NEMATODES OF DOUBTFUL PATHOGENESIS

In addition to the foregoing there are a few worms parasitic in man which have some claim to be considered tropical parasites. These are *Tridontophorus diminutus, Lagochilascaris minor,* and *Physaloptera mordens.* Owing to space considerations, they will not be considered in detail here. Those interested may consult the references given on p. 808.

G. GNATHOSTOMA SIAMENSE

The female worm only is known and measures 9 mm. by 1 mm. The anterior third of the body is set with spines. This parasite was first found in the breast of a young Siamese woman. The patient had a hard painful swelling in her breast accompanied by fever. On the skin appeared nodules the size of a bean, from one of which the worm was removed. Two other similar cases have since been reported. Similar species live in the stomach of cats, dogs, pigs, oxen, and monkeys in the Orient and in South America.

The treatment is surgical.

H. LEECHES

A number of species of these animals attack man in tropical countries. One form collected by me in West Africa is often found on dry land at great distances from water. A few of the most important tropical species are *Limnatis nilotica, L. mysomelas, L. granulosa, Hæmadipsa ceylonica,* etc.

L. nilotica is usually taken into the mouth with drinking water, or attacks persons bathing. It has been observed in the pharynx, larynx, esophagus, nasal cavities, vagina, and conjunctiva. *H. ceylonica* clings to leaves or twigs and springs upon passing men or animals. Its bites are said to be extremely liable to become painful and dangerous ulcers. Death has been known to result from the attacks of large numbers of these parasites. It should not be forgotten that leeches are the hosts of trypanosomes and other parasites, and their bite may introduce these into man as well as into animals. Numerous undescribed species occur in various parts of the tropics.

Remedial Measures

When the animals were in the nares Manson succeeded in removing them by attaching, through a speculum, a spring forceps to their hinder end, and afterward injecting salt and water. They should be carefully looked for in the mucous orifices of the body when their presence is suspected. When attacking the skin they should be promptly removed and an antiseptic applied.

Preventive Measures

It is sometimes difficult to protect one's self from attack, as some species are able to force their way even under close-fitting garments. Manson recommends that when passing through jungle lands in which leeches abound one should have the feet and legs protected. Care in the selection of drinking water is, of course, an obvious precaution.

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CHAPTER VI

DISEASES DUE TO BACTERIA

1. LEPROSY

PAUL G. WOOLLEY

Leprosy is an infectious and possibly also a contagious disease, chronic in its course, and cosmopolitan in its distribution. It is the expression of invasion of the body by *Bacillus lepræ,* and is characterized by localized granulomatous lesions in all the organs and tissues of the body. There is, however, a predominant tendency to involvement of the skin, mucous membranes, and peripheral nerves. The clinical types of the disease are the result of such special localization; that is to say, the tuberculous or nodular type depends upon cutaneous localization, while the maculo-anesthetic type is the result of invasion of the sheaths of the peripheral nerves. The two types may be combined. Ehlers suggests that the anesthetic form bears the same relation to the tubercular form that tabes dorsalis does to secondary syphilis.

The prognosis, while not exclusively bad, is almost uniformly so. There have been some few cases reported as cured, but, if one considers the extreme chronicity of the disease, the extremely protracted incubation period (from 2-32 years), as well as the fact that superficial lesions may disappear spontaneously or be caused to disappear without affecting in any way the course of the disease, it becomes evident that in order to determine completely recovery it must be shown that no lesions, or at least only healed ones, exist in the body. Few, if any, case reports satisfy such a requirement.

All of this is especially interesting in view of the fact that leprosy is one of the most ancient of known diseases. When one considers that nearly sixty centuries of empiric treatment have given practically no results, so far as cure is concerned, one realizes the hopelessness of further experiments that do not start from fundamental biologic facts. The time for logical experimentation in the therapeusis of leprosy has only just arrived, and the future gives hope of great and perhaps complete success.

There is an excellent explanation for the backwardness of our knowledge of leprosy, in the fact that in this disease, as in few others, prejudice based upon ancient authority and superstition prevented careful studies of the means, methods, and laws of transmission. So long as lepers were objects of loathing and of fear, and so long as their disease was believed to be the result of the vengeance of demons or spirits or gods, complete study or even thorough study was impossible. But, since the loss of supernatural interpretations, and especially since the discovery of the causative organism by Hansen (1871), more and more energy has been devoted to the study of leprosy by larger and larger numbers of workers. The attempt to discover the facts in the life history of the organism has been the chief point of attack. Only within the past two years has important fruit been borne of these efforts. Clegg, Duval, Duval and Gurd, Campana, Sugai and Nicolle, and Blaizot have succeeded in obtaining cultures of B. lepræ outside of the human body and in producing lesions in experimental animals with the organism. We may now hope to discover something definite concerning the portals of infection and the methods of transmission in leprosy.

Etiology.—Leprosy, it has been assumed from time immemorial, is a "filth disease." It is more frequent among the poverty stricken, dirty classes of the population. It decreases whenever hygienic conditions improve. This suggests that constitution and disposition are unimportant as compared with exposition. Such a view would offer an explanation of house infection and the so-called racial immunity to leprosy.

Dark-skinned races seem to show a greater leprosy incidence than the white races, but it is also true that the hygienic standards of the dark races are not as high, generally speaking, as those of the white races. Nevertheless, creolization modifies the incidence so that the disease is less frequent among mixed black-white races than in the black, but more frequent than in the white.

Leprosy is not an hereditary disease. No undoubted case of fetal leprosy has been reported. But it is not improbable that congenital infection may be frequent, though not extremely so. In India the juvenile cases varied from 4-8 per cent. (Azoulay). In other parts of the world they rarely exceed 3 per cent.

If we concede that leprosy is an infectious disease, then, indeed, we must believe that the human being is, as a rule, quite resistant, for wherever the disease exists the opportunities for infection are legion. The bacilli are given off from the body in all the excretions and secretions. They have been demonstrated in the discharges of leprous sores; in the pus of pustules of lepers; in the nasal secretion; in the sputum, when there have been active lesions in the mouth, pharynx, larynx, or lungs; in the feces, tears, and vaginal secretions; in the urethra and in the seminal fluid, in the milk and sweat; and in masses of desquamated epithelium. In oriental countries, where leprosy is encountered daily upon the streets, there must be myriad opportunities for respiratory or for cutaneous infection; yet such infection is very uncommon, at least in white men.

Ordinarily leprosy bacilli are not found in the blood, but in occasional cases manifesting acute febrile reaction, due apparently to processes connected with dissemination and metastasis, they have been found free in the plasma, or included in white or red blood cells. It is an interesting, though not surprising, fact that the secretions of tubercular cases contain the bacilli more frequently and in larger numbers than do those of anesthetic cases. Sticker says that, in his series of 153 cases, 127, or 83 per cent., had bacilli in their nasal secretions. In this series there were 57 cases of nodular leprosy, of which 55 showed bacilli; 68 cases of nerve leprosy, of which 45 showed bacilli; and 28 cases of mixed leprosy, of which 27 showed bacilli (cf. Scheube). It is also true that Kitasato has found leprosy bacilli upon the nasal mucous membrane of a non-leprous wife of a leprous man, an observation which hints at the possibility that healthy persons may serve as carriers.

Obviously, then, the opportunities for transmission are many, and the possibilities as to methods of transmission are also numerous. The most anciently accepted idea was that transmission could take place through the air, and that contact with a leper was certain to result in infection. The current belief is that transmission results only from direct or indirect personal contact, and that cutaneous wounds offer advantages in this regard. Peterson thinks that skin lesions of any sort favor infection, and some authors go so far as to suggest that the organisms may penetrate the unbroken skin by way of the hair follicles, the sweat or the sebaceous glands. Transmission by way of the respiratory tract has been suggested also, but, if we exclude the nasal passages, it is probably excessively infrequent. If we include nasal infection, it is perhaps the most probable method. Sticker believes that leprosy, like glanders, is, as a rule, primary in the nasal cavities. It is suggested that infection is brought about by bacilli in the fine spray produced by speaking, coughing, or sneezing by an individual who has leprous lesions in the respiratory tract or mouth. Certainly the prevalence of bacilli in the nasal secretion favors this method. Infection by the gastrointestinal tract has been suggested, probably because of splenic and hepatic localization of lesions. Such a method of transmission might result from food infected in one or another way. It is, however, more probable that splanchnic localization is the result of swallowing lepra bacilli with the secretions of the upper air passages.

Transmission of leprosy by sexual contact also has been suspected as a possible means of spreading infection. Thiroux found that 27.27 per cent. of the women with nodular leprosy and 3.8 per cent. of those with the anesthetic type, whom he examined, harbored lepra bacilli in their vaginal secretions.

Also leprous infection by vaccination has been reported (Gairdner and Daubler).

Aside from these suspected direct and indirect personal methods, another means is possible, namely, transmission by insects. And, because of the commonly unhygienic conditions of life among the persons affected with the disease, this method has received considerable attention. Pediculi, pulices, acarids, cimices, culicidæ, tabinidæ, philodromes, simulia—all have been suspected of being implicated in transmission. With regard to all of these we may safely conclude that, if any of them do play any part in transmission, it is a purely passive part, the observation of Goodhue and Taylor and Noc to the contrary notwithstanding.

So far, then, as transmission is concerned, all that we know leads to the conclusion that leprosy is a disease that is propagated by intimate social contact.

Concerning the occurrence of the bacilli outside of the human body we know nothing. It may be possible that in the future we shall discover some close relationship between rat and cat leprosy and the human form, but for the present it seems barely possible that the bacilli of animal leprosy are only related to human leprosy as the bacilli of animal tuberculosis are to the human disease.

PROPHYLAXIS

Consideration of the foregoing facts—i. e., that portals of infection and the methods of transmission of leprosy are not known; that it is associated with unhygienic social conditions; that the cause of the disease may be present in all the secretions of the body; that healthy individuals associated with lepers may act as carriers—leads to the conclusion that the only complete prophylactic method of dealing with the disease is the ancient method of segregation. In its earliest stage this amounted to ostracism; later, in the time of the crusades, it was ostracism plus voluntary segregation; now it is, in its best form, isolation by colonization. In whatever form the method has been applied it has been successful, but it has not been as widely applied as it should be because of the coercive aspect which has always been associated with it. Coercion will be almost unnecessary when colonies or sanatoria are made as attractive as possible; when leprous families can live their own lives in reasonable privacy; and where there is congenial work for those able to work. But first, in order to make segregation possible, registration should be compulsory, and, in localities where segregation cannot be enforced, quarantine is very useful.

Much could be done in a general way by educational methods in accomplishing hygienic reforms. Hygienic standards of living should be improved, and if it were possible to provide separate dwellings for separate families, and so ameliorate overcrowding, a most important advance would be made.

Whether or not segregation is practiced, the regulations of both public and personal hygiene should be carefully enforced.

All of the secretions of patients, especially those with the nodular form of the disease, should be carefully disinfected. Dressings should be burned, clothes, towels, bedding, and utensils should be boiled or otherwise sterilized, and patients should be kept free from insects. The skin should be kept clean, and cutaneous activity should be promoted by baths, medicated or non-medicated.

The mental life of the patients should be provided for. Suitable work should be provided for them, amusements should be encouraged, and families should not be separated. From the fact that so few children born of leprous individuals become lepers, even when they remain associated with their parents, various authorities and, indeed, the Second International Conference on Leprosy (vide infra) have recommended permitting lepers to marry, provided the individuals remain in segregation, and provided that the children resulting from such marriages are removed from the colony after birth.

The question of disposal of the dead seems important. Arning states that apparently viable lepra bacilli have been found 5 months after burial. If this be true, then cremation should be made compulsory, or at least bodies should be completely surrounded by lime and buried deep in the soil. Duval and Gurd comment upon the viability of *B. lepræ* and upon, its retention of dangerous qualities, and insist upon the danger of lack of attention to the possibilities of infection from secretion. Leprasoria, therefore, should be kept in the rigidly sanitary condition, and dwelling houses should be subject to careful inspection and disinfection.

TREATMENT

Concerning the treatment of leprosy there is really very little to be said. It is possible that the next few years will change all this. For hundreds of years the empiric method has been in vogue, and for good reasons. Only perhaps in very early times, when the disease was looked upon as a result of the malign influence of evil spirits, was it treated logically—that is to say, by exorcism. Later, when demons were abolished, the purely empiric method was applied so persistently that, if one considers the various things used by oriental medicine men, few substances have been untried. And all to no avail.

It would consume far too much time and space to discuss in detail the various sorts of treatment that have been used or which are being used in the treatment of leprosy. Because of the inadequacy of all treatment it almost suffices to say that there is no specific treatment, and that true causal therapy has not had time to be developed. Carrasquilla has experimented with a serum obtained from horses which had received injections of leprous blood, and Herman and Abraham have used a similar serum obtained after injecting horses with the tissue juices of lepers; but both failed to obtain satisfactory results. It was claimed that Rost's leprolin was obtained from cultures of the leprosy bacillus. Rost's methods have been severely criticized and the product has little, if any, value. Castellani and Woolley have each tried autogenous vaccines prepared by making emulsions of the bacilli obtained from the lesions of lepers. Recently Whitmore and Clegg made experiments with vaccine produced from cultures. All were without success.

Concerning treatments other than specific or causal, there is little to be said, save that they have no immediate effect upon the disease, but that any useful method of treatment has merely an individual effect because of improvement in the general condition of the patient. Treatment is, therefore, almost entirely symptomatic, supportive, tonic, and hygienic. Of these the hygienic is as useful as any. The disease has periods of spontaneous improvement, and one cannot be certain whether the treatment is always the important factor. Therapeusis is, therefore, delusive. Jonathan Hutchinson reports a case in which apparent recovery could be ascribed to nothing else than port wine! Moreover, Pernet says that mild cases may be prevented from becoming serious, and that they may be cured, by hygienic methods alone. When a case has become frankly nodular—when the typical facies has developed—treatment can achieve little but physical relief, and a therapeutic cure is hopeless.

However that may be, innumerable drugs have been tried, and many of them persistently. Antimony, mercury, arsenic, phosphorus, sulphur, iodin, carbolic acid, creosote, iodoform, salicylates, formalin, etc., and even Valentine's meat juice (subcutaneously), tuberculin, thyroid extract, and antivenin have been tried. Medicated baths, containing iron and arsenic, permanganates, etc., have been experimented with. Finally, all sorts of vegetable preparations and substances extracted from closely related or apparently closely related bacterial growths.

At the present time two substances have attracted most favor. These are Chaulmoogra oil and Nastin. Chaulmoogra oil is obtained from seeds of *Gynocardia odorata* (*Lindlei*). It is used both internally and externally. For internal medication the oil is given thrice daily in 5-drop doses at the time of onset, gradually increasing to 30-50 drops or even to 20 grams. It has a tendency to disturb the stomach, and hence is given in milk or capsules after meals. Externally it is used, diluted with bland oil (1-10-20), as an inunction, and rubbed thoroughly into the skin twice daily. It may also be used in the form of a clyster, for which it is diluted with milk (12-75), or as a hypodermic injection (5.0).

A purified form of oleum gynocardiæ (Chaulmoogra) passes under the name *antileprol.* It is a neutral oil and seems to be less objectionable than Chaulmoogra oil, because it produces no gastric disturbances and because it causes no reaction when used hypodermatically.

Neither of these oils, however, produces other results than a more or less uncertain improvement in the general condition of the patient. So far as is known no cure has been effected. The beneficial general influences of Chaulmoogra oil are said to result because of increased perspiration, improvement of appetite, increased suppleness of the skin, decreased anesthesia, and decrease in the number and size of the nodules. Such good results also follow systematic bath treatments, such as those of Kusatsu in Japan.

The second popular substance of the present time is *nastin.* This substance is a bacterial fat produced by a species of streptothrix which Deycke and Reschad isolated from the lesions of a severe case of leprosy. According to Deycke, only the unchanged nastin molecule is active, since no results are obtained with the separated fatty acid or soaps. Pure nastin produces very marked reactions in leprous tissues, characterized by inflammatory swelling, purulent softening, and necrosis. In combination with benzoylchlorid, however, the reaction is either absent or very slow in development. Hence, the use of diluted mixtures of the active substance in benzoylchlorid. Nastin B is equivalent to 0.05 per cent. nastin; Nastin B_2, to 0.2 per cent. nastin.

Treatment consists in subcutaneous injections of 1 c. c. of Nastin B once a week from 5-6 weeks, and this is followed by a series of similar doses of Nastin B_2. The general results are that in a certain number of cases the general condition of the patient is improved, and in a few the leprous process improves. The literature seems to be about equally divided between the opponents and proponents.

With reference to these fats and oils, the question has been asked whether persistent use of any non-irritating oil would accomplish similar results (Abraham).

The hygienic treatment, as has already been indicated, is important. Leprous individuals should first of all be kept contented. Work and play will do much to accomplish that result. Avoidance of direct sunlight will assist, for strong sunlight is said to increase the pain in exposed lesions, to increase the development of lesions, and to aggravate the general condition. Exercise is useful, especially if taken in the cool part of the day and in the shade, and in the absence of flies and mosquitoes, which are irritating.

The food should be easily digestible and of good quality. Spices and acids are suspected of producing unfavorable results. Meat and fish are permitted if they are fresh (Duque), though Hutchinson says that without abstinence from fish recovery is almost impossible.

With reference to other treatments, there is little enough to be said. Mercury has been used in every conceivable way to no effect, save when syphilis existed in combination with the leprosy. Arsenic also has been repeatedly tried, and seems to be the most satisfactory mineral agent. Danielssen reports excellent results with it in combination with sodium salicylate, cod liver oil, and quinin.

Equally good results have been reported in some cases with the Röntgen rays, with radium emanations, and with Finsen light. These results have, however, been no more than superficial improvements. For cosmetic purposes these agents, or antiseptic solutions, as well as ichthyol, resorcin, pyrogallol, chrysarobin, oil of Cashew, are all useful in individual cases. Europhen, formalin, salicylic acid, and chinosol have also been used with varying success.

Surgical procedures are often desirable for cosmetic purposes. Wounds heal well, as a rule, in leprous patients, because, as has been suggested, there is decreased sensibility of the nervous apparatus. Amputations may be done when necessary, and they frequently do much general good, especially when the tissues are sloughing and the danger of absorption of toxic products exists.

Occasionally nerve-stretching is useful in the relief of pain. It has been said that this procedure exercises a beneficial result in perforating ulcers.

Troublesome cutaneous nodules may be excised; necrotic bones removed; incisions made to improve contractions; tracheotomy to prevent death from asphyxiation due to laryngeal lesions.

2. PLAGUE

WILLIAM B. WHERRY

PROPHYLAXIS

Comparatively recent discoveries have placed in the hands of man a sure remedy against the plague. For now we know the elements which, when brought into conjunction, start that prairie fire which thrice, in recent times, has swept the earth with its destroying blast. Certain rodents, their fleas, and man are the combustibles; *B. pestis* the spark.

True, conservative medical prophylaxis looks into the future as far as possible and builds a barrier which, though like the wall of China it takes a hundred years, stands forever. All efforts which bring about temporary prophylaxis alone are wasted along with time and money.

Why is it that even so-called civilized races resist our efforts to bring about immunity to disease? The answer would be totally discouraging were not ultimate victory so desirable. It may take centuries and require marked changes in our social conditions to bring about the real prophylaxis of plague; and there are many who believe that the carrying out of permanent prophylaxis to completion cannot be hoped for excepting under a centrally organized bureau framed after the fashion of that so beautifully presented by Osler in "The Nation and the Tropics."

Long ago mankind associated death among rats with the epidemic prevalence of plague. Perhaps this popular belief antedates those on the relation between mosquitoes and malaria; the tsetse fly and its diseases; stagnant water and the Medina worm. However, it took the extensive experiments of the last Indian Commission to convince us that rats and their fleas are the chief factors worth considering in the epidemiology of bubonic plague. These experiments lend more than ample support to those of Simond, Gauthier and Raybaud, Verjbitski, and others, and have been confirmed in Japan by Kitasato.

The observation of Liston that guinea-pigs may be carried off by an epizoötic of plague was of great value in solving the problem of transmission. Numerous experiments showed that feeding, grossly contaminated surroundings, direct contact with the sick, and infected air utterly failed to start an epizoötic, provided fleas were excluded. Healthy rats, guinea-pigs, and monkeys, exposed to infection in plague houses, died of the disease; and rat fleas containing plague bacilli were found upon them. When such animals were suspended above the floor higher than a rat flea can jump (about four inches), or protected by a strip of "tanglefoot" fly paper six inches wide, they remained free of fleas and escaped infection. Plague-infested fleas could be picked off the tanglefoot. Controls not so protected or only two inches off the floor were attacked by plague-infested fleas and subsequently manifested the disease. It was shown that a flea may take in as many as five hundred plague bacilli from the peripheral blood of a rat dying of septicemic plague; that the bacilli multiply in the flea and are discharged in a virulent state with its feces; that a flea bite is a sufficient atrium of infection, when such material is rubbed into it, or simply deposited upon it. While it is possible that a single infested flea may be successful in transmitting the disease, the chances are increased in direct proportion to the number of infected fleas which attack a susceptible animal. A prevalent objection was set aside by demonstrating that rat fleas, when starved, are not particular about the source of their next meal; that they willingly bite man and other animals. However, when they get on man, they take some time to feed on him, and this is of importance in connection with the rôle which man plays as a carrier of infection from one place to another. Rat fleas, when starved, do not live longer than a week, but may be kept alive for three to four weeks on human blood alone.

The Relation of Epizoötic Rat Plague to the Human Epidemic.—This was demonstrated in the epidemiologic observations in Bombay and in villages in the Punjab. The substitution of a guinea-pig population for a human one in deserted villages lent the character and accuracy of a laboratory experiment. In every instance the outbreak in man or guinea-pigs was preceded by an epizoötic in rats.

The Species of Rodents and Fleas Concerned.—In Bombay an epizootic in the Norway rat, *Mus norvegicus* (*decumanus*), preceded that in *M. rattus,* which in turn antedated that in man. In the Punjab, *M. rattus,* being the most numerous, played the most important rôle. The rat fleas, *Xenopsylla cheopis* and *Ceratophyllus fasciatus,* and the human flea, *Pulex irritans,* all transmitted plague experimentally; but in India the first species is chiefly to blame. In America *M. norvegicus,* being by far the commonest species of rat, is most often infected and carries a relatively greater number of *Ceratophyllus fasciatus,* the flea with which Gautier and Raybaud first performed successful transmission. Experiments with the cat and dog fleas have been unsuccessful.

The Relation of Other Animals to the Spread of Plague.—The earlier beliefs in the susceptibility of man's domestic animals to plague were shattered by the extensive experiments of Hill, and Bannerman and Kapadia. Among rodents, plague is known to occur naturally in the cosmopolitan Norway rat (*Mus norvegicus*), black rat (*M. rattus*), Egyptian roof rat (*M. rattus alexandrinus*), and mouse (*M. musculus*); also in the *Nesokia bengalensis*—small bandicoots in India; the tree squirrel (*Sciurus palmarum*) in India; the tarbagan or tarabagan (*Arctomys bobac*), a ground squirrel in Siberia and the Himalayas (Barykin); the guinea-pig (Liston); the California ground squirrel (*O. beecheyi*) (Wherry); the brush rat (*Neotoma fuscipes*) (McCoy). In North America the following animals have been found to be susceptible to inoculation: the rock squirrel, desert wood rat, prairie dog, weazel, chipmunk, and pocket gopher (McCoy); in South America the llama (Biffi). According to J. A. Thompson, marsupials acquire it in Australia. Several species of monkeys, which associate with humans in India, have acquired it naturally. Cats may develop a chronic form with cervical buboes. Bats have been shown to be susceptible to inoculation. During an outbreak of plague in Eng-Chhun, Fu-Kien province, China (1900), Dr. Philpot Lawrence noted that the bats died in large numbers of a plague-like disease (personal communication). In judging the relative importance of these animals, so far as the epidemiology of plague in man is concerned, one must consider the intimacy of their contact with man and their relative susceptibility. On this basis rats and mice take the first place. The important rôle played by ground squirrels will be referred to later.

The Relation of Man to the Spread of Plague.—The contagious nature of primary pneumonic plague has not been seriously questioned, for no frank outbreak of this form of the disease has occurred during the bacteriologic era, until it appeared in Manchuria (1910-11). The cold weather and crowding indoors apparently favored its rapid transmission from person to person, and often whole households were wiped out. According to Haffkine and the Japanese bacteriologists, it was not accompanied by plague in rats (Gray). Perhaps the degree of terminal septicemia here does not allow of its transfer to rats. Man, as compared with the rat, is not a very favorable source of infestation for the flea. The blood of a rat dying of plague septicemia may contain from a few thousand to a hundred million or a billion plague bacilli per cubic centimeter, while that of man rarely contains over a few thousand. Man, however, may transport the bubonic and septicemic type by traveling while ill, and acting as a source of infestation for fleas; or, as the healthy carrier of clothing, etc., harboring plague-infested fleas, which, subsequently escaping, start a new focus of epizoötic among rodents.

The Relation of Unsanitary Conditions to the Spread of Plague.—The only conditions which influence the origin or spread of bubonic plague are such as concern the food supply of rats and such as furnish them harboring and breeding places: the improper disposal of garbage; the careless storage of meats, grains, and other foodstuffs; the presence of rat runways through defective foundations, floors, and roofs, and along unsealed plumbing. The relative immunity acquired by Europe, North America, and certain other localities to this *miseriæ morbus* is undoubtedly due to the gradual improvement made in public hygiene. In the case of pneumonic plague, overcrowding, as in the poor districts of cities, furnishes the close contact apparently necessary for transmission.

The Seasonal Prevalence of Plague, and the Factors Influencing It.—It is an old observation that certain meteorological conditions influence the spread of plague. In cold and temperate regions epidemics usually occurred in summer, whereas in hot and dry regions, central Egypt, Mesopotamia, central India, etc., the disease died out in summer. The English Commission give the only logical explanation offered, and base it on the number and susceptibility of rats; observations on the bionomics of fleas; the influence of temperature on the fate of the plague bacillus in the stomach of the flea; the influence of temperature on the grade of septicemia developed by rats before death.

The most important factor concerns the number and susceptibility of the rats. They greatly outnumber the human population. In equable or warm climates they breed throughout the year. Extremes of heat and cold retard multiplication. The character and amount of food supply further modify the rate, which is naturally increased when their numbers are temporarily diminished, as by an epizoötic of plague. The English

Commission found a relative decrease at the close of the epidemic season in Bombay, and noted that this was followed by accelerated reproduction. In Bombay 59 per cent. of the rats were found to be immune to relatively large doses of plague bacilli. Two years later they exhibited the same degree of immunity. McCoy found 50 per cent. of large and 15 per cent. of small San Francisco rats to be immune long after the epidemic had passed. This immunity is naturally acquired by age, and is probably analogous to that acquired by rats to *B. pestis caviæ,* as shown by the writer.

Rat fleas are actually much more numerous during the epidemic than during the non-epidemic season. In India the high temperature of the off season not only interfered with the deposition of eggs, but restrained the eggs from developing into larvæ.

A mean temperature of 85° F. and over favored the rapid disappearance of plague bacilli from the intestinal tract of the flea. This clearing was attributable mainly to phagocytosis—the flea in its subsequent feedings taking in the necessary opsonins and leukocytes, which for their action require a suitable temperature. The period of infectivity of plague-infested fleas varied with the season, being six times greater during the epidemic than the non-epidemic season.

The degree of the terminal septicemia in a rat was found to be greater at 70° F. than at 50° F.

In India the above factors operate favorably in the hottest season only in certain localities, where sporadic rat and human plague bridge over the non-epidemic interval.

The Recrudescence of Plague.—One may well inquire whether other factors than those hitherto considered may not really be of greater importance in favoring the persistence of plague on the earth. Will assuming the rat to be the original host of *B. pestis,* and the sporadic occurrence of plague in rats and men, explain its persistence throughout the centuries, and between the great pandemics? It is not possible to answer this definitely for the world, but, so far as America is concerned, the most favorable host for the plague bacillus has been found among the *Arctomyinæ.* Since the writer (1908) proved the current belief that plague existed among the ground squirrels of California, all the evidence confirms his statement that, "altogether likely, ground squirrels may act as a host for the *Bacillus pestis* in the interim between the more noticeable outbreaks in rats and men." While absent (1908-11) among rats and men, excepting cases of squirrel origin, plague has been shown by Blue, McCoy, Rucker, and others to persist over a widely scattered area in the California ground squirrel; and these rodents have apparently maintained the infection since its introduction in 1900. It is significant that, at least in one other "Endemic Center" in the transbaikal region of eastern Siberia and northern Mongolia, between 50° and 54° N., plague is known to exist in a related rodent, the tarbagan (*Arctomys bobac*). Attention was first directed to this center in 1895 by the Russian doctors Bjaljawski and Reschetnikow. In 1900 Rudenko presented epidemiologic and clinical observations, which left little doubt that the tarbagan disease so often transmitted to man was plague. Following the demands of the medical press for bacteriologic proof, the Russian government sent Dr. Schreiber, who later died of plague, to the infected region. At the end of 1905 and in the beginning of 1906 two expeditions were made, the latter covering one thousand miles, but no cases were seen, for apparently the disease only occurs in the autumn. Schreiber collected and published data of previous outbreaks. The bacteriologic evidence was partially supplied by Barykin, who in the autumn of 1906 and 1907 noted a mortality among the tarbagans near Manchuria Station, and this was followed by pneumonic plague in humans. He found one tarbagan with a swollen spleen containing bacteria, which microscopically resembled *B. pestis*. His clinical observations and bacteriologic investigations on cases in man, where infection was acquired in the hunting or skinning of tarbagans, prove that the disease was plague. Every summer the population in the neighborhood of Manchuria Station is greatly augmented by Chinese, who flock in to hunt the tarbagan. It was among these men that the recent epidemic of pneumonic plague originated and thence spread eastward to the sea.

LeDantec has looked upon the tarbagans as the permanent source of plague in rats and men, and has urged the primary necessity of waging war against them. But it must be conceded that, were it not for rats, plague among the Arctomyinæ would be of little significance, owing to their less intimate contact with man.

The Extermination of the Rat.—Thus the plague problem is narrowed down to the Rat Problem, so well presented by Boelter.

Rats did *not* embark with Noah. For is it not revealed in the *Quassul Ambia* of the Mohammedans how that patriarch, having forgotten in his hurry to install sanitary arrangements in the Ark, appealed for help from on high. The pig was created to clean up the accumulated offal; but the ever-restless Shaitan drew forth from the pig rats, which multiplied enormously, and their gnawings endangered the whole animal kingdom. The angel Gabriel, descending upon request, instructed Noah, and he, passing his hand over the nostrils of the tiger, drew forth cats, which soon held the rats in check. One should be glad to know this in order to place blame and shame where they really belong. We must admit, however, that Shaitan was clever when he endowed his children with such extraordinary fecundity. After thousands of years suffering mankind has waked up, in spots, to the moral necessity of making a concerted effort against the offspring of that Evil One. We have national and international societies for the destruction of vermin, and one small nation has found it profitable from a business standpoint alone to place a bounty on rats. This is a good beginning, but there seems to be some confusion about the *modus operandi.* Had those Poles not checked those Eastern hordes, we would have taken in with our mother's milk the knowledge that the cat is the especially created enemy of the rat, and the reasons therefore, and so have averted the just criticisms of Buchanan. To expect extermination of the rat seems preposterous, for we are but human. However, so far as plague prophylaxis is concerned, that is unnecessary; the factor of safety may be reached by reducing their numbers. For this the reasons are now evident.

Buchanan prevented the recurrence of plague in certain Punjab villages by importing cats. His observation that villages with 30-75 cats per hundred houses escaped plague, while those with a low percentage were attacked, are noteworthy, and should lead to extensive experiments in a land where ratproofing is hopeless, and where religious prejudices interfere with wholesale rat destruction. "It is one of the measures of plague prevention dictated by their scriptures to Mohammedans and Hindus alike, and which will, therefore, be acceptable to all."

Independently, R. Koch expressed the opinion that the only solution lay in the breeding and maintenance of an efficient race of cats. Like Noah, he found that keeping them on ships bound for the tropics insured comparative freedom from rats. His plan has been advocated by Kitasato in Japan. There the latter found that the percentage of cats to houses varied from four in Tokio to forty-nine in the Yamanashi district, where cats are kept to protect the silk industry from rats. Shiga considers the latter place safe from plague; we know that the former is not. That sounds like plain sailing, but Shaitan has kept busy—he put the *Cysticercus fasciolaris* in the livers of rats and mice so that cats may suffer and often die; and strange ideas into the brains of the more weak-minded humans.

The only other rational plan of attack is aimed at the rats' food supply. Like any other living thing, they multiply up to the limit of their food supply, and any prophylactic measures which leave this fundamental fact out of consideration must have an evanescent effect. The most persistent trapping and poisoning yields only very temporary reduction. Wherever careful census has been taken, it is noted that the primary diminution in numbers favors increased multiplication; and, other things being equal, the rat population soon regains its former strength. After four and a half million rats had been killed in Tokio, they were found to be still numerous. Ratproofing, in order to be efficient, must be carried out conscientiously over a long period of time, and, at the very least, in all ports receiving foreign shipping. In America this would be insufficient, for we already have an endemic focus of plague. Lands still free of infection may well reflect on the fact that, in spite of modern quarantine regulations, including inspection and disinfection of ships and rat guarding of ships' cables, the present pandemic has invaded fifty-two countries.

There are no reasonable or insurmountable objections to this plan of attack, unless they exist in present social conditions. It is the easiest thing in the world to make a building ratproof, and the hardest to get anyone to do it. When a corporation or large stable owner is able to successfully evade an explicit ratproofing ordinance in the face of a State Board of Health backed by the Federal Government, one must surely conclude that the relation of hygiene to material progress is still unappreciated.

Measures Yielding Temporary Protection.—1. *Inoculation.*—In the meantime, where social conditions permit, among the simple-minded, so-called savages; among physicians, nurses, and sanitary inspectors, and in such others to whom perpetuity seems desirable, one may produce temporary immunity to plague by vaccination with dead or living attenuated cultures of *B. pestis.* The carefully collected statistics of Bannerman, based on hundreds of thousands of inoculations in India, prove the protective powers of Haffkine's prophylactic. Not only is the incidence reduced, but also the case mortality among those inoculated.

Undhera, a village of farmers near Baroda City, was swept by plague in 1898. Between January 5 and February 12 seventy-six persons died of plague, ten had left the village, and four were born, thus leaving 950 persons alive—giving a mortality equivalent to a death rate of *766 per mille per annum.* The men, women, and children in each hut were divided into two groups, inoculated and uninoculated. Six weeks later, when plague had ceased, a census was taken again, with results so striking and pathetic as never to be forgotten by the plague officials. In one instance, from two huts in succession, only the inoculated answered to their names. All those untreated had died of plague. Altogether there had been 8 cases with 3 deaths among the inoculated, and 27 cases with 26 deaths among those untreated. If the uninoculated had been injected their mortality would have been reduced 89.6 per cent.

In a record of over 6,000 attacks of plague among the inoculated the mortality was 43.5 per cent., against 73.3 per cent. among the uninoculated. Of the total, 358 developed plague one to ten days after inoculation, and of these 172 died—a mortality of 48 per cent. Many figures like the last refute the contention of Calmette, that one's chances are diminished by inoculation during the incubation period.

The prophylactic consists of a six-weeks-old broth culture of *B. pestis* killed by heating at 55° C. for 15 minutes and preserved with 0.5 per cent. carbolic acid. The method of bottling and control instituted by Bannerman in 1905 absolutely rules out the repetition of such an accident from tetanus as occurred in Mulkowal in 1902. The dose for an average healthy man is 5 c. c. injected subcutaneously in the back of the upper arm, which is previously sterilized with 5 per cent. carbolic solution. Children may be given relatively large doses. Persons over 50 should get one-tenth less than the adult dose for each decade above that age. Pregnant women may be inoculated up to the seventh month without special reduction, but at later term the dose should be divided in two with a week's interval. Miscarriage has never been known to follow inoculation. This is fortunate, considering the fatality of plague during pregnancy.

Those suffering from other fevers should not be inoculated until forty-eight hours after the fever has entirely gone.

In serial inoculations the syringe is best sterilized by Wright's method: it is simply filled with oil heated to 160° C. (320° F.) and emptied at once. Between each operation the needle is simply dipped into the oil.

In three to five hours after inoculation there is swelling and pain at the site of inoculation, a rise of temperature, and general discomfort. No treatment is required, excepting ice to the head, if there is headache. No change in diet or occupation are necessary. The general symptoms usually subside in twenty-four to thirty-six hours; if not, a purge will give relief.

Shiga's modification may be of use when a vaccine must be prepared in a hurry. It consists essentially of a similarly heated and preserved physiologic salt solution suspension of *B. pestis* in the proportion of one oese (2 mg.) of *B. pestis* to 1 c. c. of salt solution. For the first injection 0.6 c. c. is given, and when symptoms have subsided another inoculation of 1 c. c.

Strong believes that inoculation with living avirulent cultures of *B. pestis* yields greater and more lasting immunity. The operation of this method on a large scale would seem to present many difficulties and dangers.

The duration of immunity conferred by the Haffkine prophylactic has been proven to be longer than at first believed. From a searching inquiry into the causes of death, during five years among the Parsis of Bombay, "it would appear that the degree of protection remaining during the second year after inoculation is only slightly less than that of the first year, and that even after five years some trace of protection remains." Some twenty villages in the Punjab passed successfully through a second plague season without reinoculation in the interval.

2. *Rat Destruction.*—By trapping, poisoning, and the use of bacterial viruses, the rat population may be reduced to the point affording temporary protection. It is labor lost, however, unless supplemented by a most thorough system of inspection leading to the proper disposal of garbage and the conservation of foodstuffs by the use of ratproof receptacles and the temporary ratproofing of stables, graineries, warehouses,

etc. Traps equaling in number at least two per cent. of the human population must be set simultaneously over the whole city or town; and the rat collections used as a guide to the work of sanitation. Since the work of the Indian Commission points to the young generation of rats as the chief source of danger, the extensive use of a bacterial virus like *B. pestis caviæ* would aid the reduction by trapping. Young rats are particularly susceptible to this organism, and sucklings become fatally infected through their mother's milk, as shown by the writer. However, it is questionable whether the use of viruses belonging to the *B. enteritidis* group is justified, excepting in the presence of acute epidemic plague.

A trustworthy rat-catching corps can only be obtained by a careful weeding out process, and then their experience should lead to permanent employment. This was the experience of Thompson at Sydney and ours on the Pacific Coast. The prophylactic value of such a permanent corps in a seaport has been demonstrated by Thompson.

3. *Measures Directed Against the Flea.*—According to L. O. Howard, one may render houses immune to fleas "by dissolving alum in the whitewash or kalsomine applied to the interior walls, putting sheets of thick paper that have been dipped in an alum solution under the floor matting, and scattering pulverized alum in all crevices where insects might lodge or breed. Powdered alum may be sprinkled upon carpets, already laid, and then brushed or swept into their meshes with no injury to the carpet, and with the certainty to many insects, including both moths and fleas. Sheets that have been soaked in alum water and then dried may profitably inclose those that are spread nearest to the sleeper."

4. *Protective Clothing.*—Sanitary officers in particular will find that, in addition to inoculation, a fleaproof suit with adjustable mask will conduce to peace of mind. Almost a century ago James Johnson wrote: "A most important measure is the use of *oiled dresses,* the texture of which is so completely close as to prevent the passage of the most minute particles of matter from without. By these means every attendant on the military pest hospitals in Malta escaped the contagion."

Measures in Vogue at Present Which Should Be Abolished.—In order to obtain the most prompt results in the control of an outbreak of *bubonic plague,* every effort should be directed against the chief sources of infection. It is evident that the detection and quarantine of cases of *pestis minor* is without the importance formerly attached to it, and that these cases are only of value as an index of the presence of plague. Likewise, the money spent in the disposal of the dead in lime in zinc-lined coffins could be more profitably used in rat destruction; ordinary coffins taken to the home of the dead plus chloroform will render the removal of the bodies infinitely safer. Since the infection does not reside on the walls or floor of the house nor in the human case, but in rats and fleas in the rat runways and cracks of the house, ordinary disinfection is useless. As a general rule, infection enters a house but once during a single outbreak, and when its presence is recognized the only measures of value are thorough fumigation with sulphur dioxid after cutting off all channels of escape for rats.

The expenses incident to the isolation and treatment of cases of the *bubonic* type in special hospitals is decidedly questionable. They will do just as well at home and are *per se* without menace to the community. The safest place during an outbreak is inside of a plague hospital; this is true even in India, as pointed out by Lamb and Liston.

The Prophylaxis of Pneumonic Plague.—In the recent epidemic in Manchuria and China the railway was the most important factor in its dissemination, and almost every town infected became so after the arrival of passengers from an infected area. Apparently fairly close contact was necessary for transmission. Chiefly coolies and the overcrowded residents of the poor districts were affected. Here the segregation of the inhabitants in small districts by means of military cordons, isolation of patients and their contacts, and disinfection when efficiently carried out was invariably followed by a fall in the death rate. All the sanitary staff, insured in the Russia and St. Petersburg companies, were inoculated and supplied with tarpaulin or linen coats, gloves, rubber or tarpaulin socks, and were obliged, when picking up the dead or sick, to wear respirators. The writer has seen a doctor, with an air of great confidence, use a double layer of loose mesh gauze as a respirator while examining a case of secondary pneumonic plague. With the aid of Providence he escaped infection.

Since Gotschlich and others have shown that convalescents from plague pneumonia may expectorate virulent germs for weeks after the temperature is normal, it is evident that such cases should be held until their sputum is no longer infectious for guinea-pigs.

TREATMENT

The "sure remedy" offered in this article is prophylaxis. Little has been accomplished in influencing the course of the disease.

1. Serum Treatment.—The discussion concerning the value of the Yersin-Roux antiplague serum has apparently been satisfactorily disposed of by Choksy. His series includes 1,081 cases treated in India during 1905-7. In this series only every alternate case was treated with the serum, the others acting as controls. Very mild cases and severely septicemic and moribund cases were excluded. The general mortality was reduced to 49.6 per cent. In cases treated during the first day of the disease the mortality was 30 per cent.; this increased to 52 per cent. among those treated on the second day, and to over 60 per cent. among those treated on the third day. The general dosage is not given, but in one case a woman of 25 years received 420 c. c. of the serum in six injections; in another a child of 7 with plague septicemia received 270 c. c. in six injections; both cases recovered.

2. The Drug Treatment.—Thomson gives an unusually favorable report on the internal administration of carbolic acid. In a series of 143 cases he had a mortality of only 30 per cent. It was given in capsules, twelve grains (0.78 gm.) every two hours, or 144 grains daily. Carboluria rarely occurred if the drug was pure and was easily controlled by omitting a couple of doses.

It is generally considered advisable to give calomel in small doses followed by a saline early in the disease. The diarrhea which occurs later may be checked with bismuth. It would seem that opium or its preparations are contraindicated, as they interfere with the elimination of toxins. Pain should be relieved by means of ice packs. The temperature is best controlled by cold spongings. Depressant coal-tar products should be avoided, as the heart is affected early in the disease. It is best supported by digitalis and strophanthus. Ice bags should be applied to the buboes, and these should not be incised until suppuration and fluctuation are manifest. It is a matter of chance if the early excision of a bubo, advocated by some, is performed soon enough. The reports of rapidly fatal septicemia following this operation would seem to condemn it. The patient should be urged to drink as much water as possible, and elimination through the kidneys promoted, and anuria prevented by Fischer's method as given toward the close of the article on cholera. Primary or secondary skin pustules should be protected by antiseptic dressings. The heart action remains weak for a long time after convalescence, and overexertion should be guarded against.

3. ASIATIC CHOLERA

William B. Wherry

Prophylaxis

The principles of prophylaxis may be divided into Communal and Individual; and yet these are intimately bound together and are based entirely upon a knowledge of the ways of transmission. Cholera is carried from one country to a distant one solely by humans—those ill or convalescent from the disease, and by "healthy carriers." In addition, for shorter distances, by streams; by infected foodstuffs which may be eaten raw or without recooking—milk, vegetables, fruits, breadstuffs, etc.; and by flies. Explosive outbreaks in large and small communities have all been traced to a contaminated water or milk supply. In fact, the sources of infection coincide with those of typhoid and recall Osler's three F's—*food, fingers,* and *flies.*

Communal Prophylaxis.—The chief sanitary regulations which promote freedom from cholera deal with a pure water and food supply. Cholera cannot spread where these exist. Here it seems worth while to call attention to the plan of Long, which aims at the total suppression of cholera in the Philippines through the installation of artesian wells.

Protection by quarantine has been a failure excepting perhaps in the case of thoroughly isolated communities, and in Germany, where an omnipotent Bureau of Health is aided by a large corps of thoroughly trained experts. There are many loopholes in any system of land or maritime quarantine which must be applied on a large scale; and one of the holes is made by the "healthy carrier." To make any system of quarantine efficient the feces of every passenger (irrespective of social and financial status) must be examined for cholera vibrios; and those infected held until bacteriologically free. This period probably varies considerably. In 1905 Gotschlich found cholera vibrios in the intestines of pilgrims returned to Tor from Mecca. Cholera had failed to appear in Mecca that year, and yet these individuals had picked up and carried the germs for a period of more than five months.

Zabolotny states that during the Russian epidemic of 1907-8 the stools of 2,440 persons who had come in contact with cholera cases were examined, and five per cent. of carriers found. In these the vibrios persisted for from a few days to two weeks, seldom longer.

According to Jakowleff, etc., in the St. Petersburg epidemic of 1908 the carriers were fairly well distributed among men, women, and children. Three classes were found: (1) Twenty-five cases in the stage of incubation. These developed symptoms after one to three days. (2) Forty "light" cases—showing mild symptoms. (3) Sixty true carriers, all of whom had solid, well-formed stools. In these the vibrios persisted for from one to ten days.

Zlatogoroff found that in over half of 225 convalescents the vibrios persisted for from fourteen to seventeen days. In one case they persisted for fifty-six days. He refers to the investigations of Kulescha, Bruloff, and Tschniknaveroff, who found that the vibrios live for a long while in the gall-bladder, and thence escape with the intestinal contents.

Possibly Roger's method of administering permanganates (vide infra) might shorten the quarantine of carriers.

Individual Prophylaxis.—One may be surrounded by cholera for months and escape if the rule to eat nothing but recently cooked foods, and to drink boiled water only, is strictly followed. The heated foods may be allowed to cool if carefully screened from flies and guarded against manual contamination. All dishes used in the preparation, serving, and storing of foods should be scalded. If possible, one's quarters should be screened against flies and their breeding places disposed of.

It is not easy to adhere to these rules when on the march or traveling in any other way through an infected district. One may then eat with one hand and fight flies with the other, while one ponders on the classic experiments of Hankin, who found cholera vibrios in sterilized milk exposed to flies in an infected district. When heated drinks are out of the question water may be rendered safe by the addition of various chemicals. The writer found that acetozone in 1-60,000 solution acting for 15 minutes killed all but the spore-bearing bacteria in Manila tap water. Alphazone is a more permanent and equally powerful peroxid. The susceptibility of the cholera vibrio to the hydrogen ion is well known. Schultze-Schultzenstein showed that, in fluids containing albumin or peptone, or both, 0.097-0.217 per cent. of hydrochloric acid will destroy the cholera vibrio in an hour. Woolley found that one per cent. of citric acid in peptone solution was fatal to the vibrio within twenty minutes. Analyses yielded him 6.86 grams of crystallized citric acid per 100 c. c. of lemon juice; or 1.37 grams per half of a large lemon. As Rogers has pointed out, in connection with an outbreak in Bengal, the drinking of permanganate solutions may have a decided prophylactic effect. The filtration of water by means of small hand pumps is unsafe, unless some filter with as close pores as the Pasteur-Chamberland "F." is used. The filter and its connections must be boiled before each operation, for, as Theobald Smith and the writer showed, bacteria grow through the pores with extreme rapidity.

Thick-skinned fruits and vegetables such as apples, oranges, mangoes, tomatoes, etc., may be eaten raw after immersion for a few seconds in scalding hot water.

The use of alcoholic beverages is to be especially condemned, for subacute or chronic gastritis, whether due to this or other causes, is most certainly an open sesame to the cholera vibrio. Other gastrointestinal disturbances, such as may be brought on by eating raw foods or by chills, are to be avoided. English practitioners in India lay stress on the importance of wearing a flannel belly band—the cholera belt—at night. No doubt it prevents diarrhea following an early morning chill.

Protection by Inoculation.—Much experimental work, as well as numerous human inoculations, seem to show that active immunity may be produced against cholera by the injection of living or dead vibrios or the autolysate of carefully killed cultures. The method was first tried by Ferran in 1885, but was perfected through the work of Haffkine, Kolle, Wassermann, Besredka, Strong, and others.

Haffkine's Method.—An agar slant culture of virulent cholera vibrios is grown at 39° for 24-36 hours; enough sterile water is added to cover the culture to one-third its height and the growth is then washed off in this water. One half c. c. of this emulsion injected subcutaneously is the average dose for an adult. The local and general reaction following the injection is rather severe and it is doubtful whether it will be submitted to generally without force. Powell, using this method in the Assam tea gardens, inoculated 5,778 coolies. Of these 27 subsequently developed cholera and 14 (51.4 per cent.) died. At the same time, among 6,549 noninoculated there were 198 cases with 124 (62.6 per cent.) deaths.

Kolle's Method.—An agar culture containing about 20 mg. of growth is suspended in 10 c. c. of physiologic salt solution and heated to 55° C. for 15 minutes; 0.5 per cent. phenol is added as a preservative. One c. c. (2 mg. of the culture) is injected subcutaneously into an adult. Murata, using Kolle's method, reported that out of 10,000 inoculated 6 became sick (mortality 42 per cent.), while out of 10,000 noninoculated 13 became sick (mortality 75 per cent.). Numerous statistics by Haffkine and others exist, but these are considered sufficiently instructive. The severe reaction produced will bar its general use, excepting in the case of troops in the field or large working forces. Even if the milder vaccine of Strong, consisting of the products of autolysis of the vibrios, is shown to be of equal protective value, so far as communities are concerned, the only permanent solution is offered by Sanitation.

Stamping Out an Epidemic.—As much coöperation on the part of the community as is possible must be obtained along the lines of prophylaxis already set forth. The influence of heated drinks is well illustrated by Salembeni. The cholera in St. Petersburg in 1908 increased and decreased, by weeks, at the following rate: 314, 2,046, 2,437, 1,403, 703, 390, 234, 124, 94. The diminution commencing with the fourth week was not due to the work of the forces of sanitation, which was poor, but was attributed solely to the placarding of all houses with directions to boil the water and the establishment of numerous booths for the free distribution of tea.

The medical inspectors should make special search for mild ambulant cases and those in the stage of premonitory diarrhea, for these might be controlled readily by the permanganate treatment. Likewise, as shown by the work of Zabolotny, all contacts must be quarantined until clinical and bacteriologic examinations prove them free of menace to others. The importance of disinfecting excreta, soiled clothes, bedding, etc., and of holding convalescents until bacteriologically free, is evident. Philoff found the vibrios to persist, in cholera feces exposed to daylight, for 19-99 days; when kept in the dark for 18-101 days.

If open wells must be used they should be disinfected by mixing potassium permanganate solution with the water until it has a decided pink color. The color will disappear in a day or two. The well should be guarded against subsequent contamination or disinfected frequently.

THE NATURE AND ACTION OF THE CHOLERA TOXINS

There are two main ideas as to the nature and origin of the poisons concerned in cholera intoxication. The original theory of Pfeiffer, that the toxic elements are intimately bound to the protoplasm of the cells and only set free upon their disintegration, is generally adhered to. It is thought by some that Metchnikoff's soluble toxin is such a freed diffusible endotoxin. The theory of Emmerich, that cholera is a nitrite and nitrous acid poisoning due to the reduction of nitrates contained in the food, would seem to be satisfactorily disposed of by the following arguments: (a) Many of the normal bacterial inhabitants of the human intestine are able to bring about the same reduction; (b) in the classic experiments of Metchnikoff young rabbits were only susceptible while suckling their mother's milk (free of nitrates), and became immune when they reached the age when they began feeding on vegetables (rich in nitrates). This argument of Salembeni's does not seem to be good. Immunity acquired with age might here coincide with the change in diet. Often it does not do so, as, for example, in the case of the immunity acquired by rats to gastrointestinal infection with members of the *B. enteritidis* group. However, (c) the chief symptoms of cholera can be produced by injecting susceptible animals with the intracellular toxins of the vibrio.

When cholera vibrios succeed in passing through the stomach they multiply, under unknown favorable conditions, in the small intestine. They remain localized there, penetrating to but a limited extent between the epithelial cells of the glandular crypts. Only rarely, when large quantities of blood have been used, have they been isolated from the peripheral circulation. Owing to their activities large quantities of water and salts are discharged into the gastrointestinal canal. This brings on the symptoms of diarrhea and vomiting. The further action of the toxins results in the desquamation and disintegration of enormous numbers of epithelial cells lining the small intestine, and these enter into the composition of the "rice water" stools which soon replace the early fecal discharges. This loss of pints or quarts of fluid in a few hours brings about an extreme dehydration of the tissues and blood; the fall in blood pressure; the small and rapid pulse; the fall in surface temperature; the shriveling of the skin; the muscular cramps; and the anuria. If the patient lives the circulation is gradually restored, the surface temperature rises, the diarrhea moderates, and urine is secreted again; or, as is often the case, collapse supervenes again or with persistent anuria come hyperpyrexia, somnolence, coma, and death.

TREATMENT

The large number of drugs recommended in the treatment of this disease is in direct proportion to their inefficiency. It is evident that any logical method of treatment must concern itself chiefly with: (1) restoring the lost water and salts, and thus reëstablishing the circulation; (2) neutralizing the cholera toxins (absorbed and unabsorbed), combined with measures which favor their excretion; and (3) the treatment of dangerous complications, the most important of which is the anuria. That it is possible to meet these requirements is due to three pieces of research work done in recent times: the production of an antitoxic serum by Metchnikoff, Roux, and Salembeni; the placing of the salt solution and the permanganate treatment of cholera on a scientific basis by Leonard Rogers; and the researches of Martin H. Fischer on nephritis.

Serum Treatment

The efficiency of the antitoxic serum prepared at the Pasteur Institute of Paris was tested by Salembeni during the cholera epidemic in St. Petersburg (1908). It was used at the height of the epidemic. In 42 treated cases (19 very severe, 10 severe, 7 medium, and 6 light) the mortality was 23.8 per cent. The official mortality returns during the same period of time was 45.6 per cent. The limited quantity of serum on hand prevented further tests. It is unfortunate that he was unable to make control tests with normal serum, as was intended, for it seems possible that similar results might have been obtained with normal serum. Salembeni's rather striking results cannot be attributed entirely to the physiologic saline with which the serum was diluted. Intravenous isotonic salines have been abolished as of little use in the treatment of cholera. Without going into the early history of their use, which has been reviewed by Rogers, it is sufficient to state that during their use Rogers had a mortality of 51.9 per cent.; and Nicholes and Andrews, in Manila, a total mortality of 43.6 per cent. Now, as Hogan and Fischer have shown (1912), salt solutions fail to permanently relieve the symptoms of shock following hemorrhage because they contain "free" water which passes out of the blood vessels. But if to such a solution there be added a colloid saturated with water, e. g., horse serum, etc., then one has a solution which, when injected, remains for the greater part within the blood vessels.

According to Albanus, etc., the antitoxic serum of Kraus reduced the mortality in 54 cases to 57.5 per cent. against 84.3 per cent. in control cases. The serum was largely diluted with salt solution. Better results seem to have followed the use of serum of Schurupow which reduced the mortality to 29.9 per cent.

Fortunately, through the careful researches of Rogers the mortality has been reduced to 24.2 per cent. by the use of hypertonic salines and oxidizing agents alone. Aided by Fischer's method of relieving anuria, one might save many otherwise fatal cases. Experiments based on Salembeni's results and on the work of Hogan and Fischer on perfusion may show the advisability of adding serum to the hypertonic salines.

Rogers' Treatment

It may be urged by some that controlling this treatment by blood pressure and specific gravity determinations makes it too difficult for use except in a well-organized hospital. While the best results are so obtained, Rogers points to the fact that Rutherford, working under unfavorable circumstances in a district hospital, lost only 23.07 per cent. of his cases (against 62.76 per cent. on the outside) by the use of intravenous hypertonic salines alone.

As the treatment is symptomatic one should watch constantly for the signs of the serious complications which may suddenly manifest themselves in the various stages of the disease.

The Stage of Premonitory Diarrhea.—There seems to be no bacteriologic evidence that cases apparently cut short in this stage would have gone on to true cholera. However, if the stools are still bile stained the diarrhea may be checked by such astringents as dilute sulphuric acid. Opium or any of its preparations should never be used. Its evident rôle in producing a state of lack of oxygen in the tissues, and so adding to the factors which produce anuria in the stage of reaction, certainly bar its use. The patient should be kept at rest in bed, given barley water in small quantities, and guarded against chills.

Judging from Rogers' results in the next stage of the disease, it would seem highly advisable to commence the treatment here with the permanganates in the place of any other medication.

The State of Copious Evacuations and Collapse.—The majority of cases come under observation in this stage. The surface temperature is lowered to 95°-96° F. or lower, while the rectal may be 99° F. The blood pressure is below 70 mm. at the wrist in most cases. Rogers has shown that in mild cases there is a loss of 35 per cent. of the fluid from the blood; in moderately severe cases 52 per cent.; in the most severe fatal cases 64 per cent. He confirmed Parke's observation that the rice water stools contain 0.5-1 per cent. of salts but very little albumin. The percentage of chlorids in the serum of some of the worst cases was reduced to as little as 0.6 per cent. This has been confirmed by Aron, who invariably found a diminution in the quantity of water and salts in the blood in this stage of the disease. Later the water of the blood is almost restored, but the salt content remains lower than normal; therefore the blood is hypotonic. Along with the failure in the circulation the kidneys stop secreting. These conditions may be relieved by replacing the water and salts lost from the blood by:

1. Rectal Injections of Normal Saline Solutions.—In comparatively mild cases when the blood pressure remains above 70 mm. from one half to one pint of saline, 90 grains to the pint (0.95 per cent. solution), may be given as high rectal enemata every two hours during the stage of evacuations, and reduced to every four hours after reaction has taken place. The blood pressure should be taken after any copious exacuations in order to meet any decided fall by more active measures. In the rectal injections three grains of calcium chlorid to the pint may be added with advantage.

2. Subcutaneous Injections.—Rogers' hypertonic solution should be injected a pint (500 c. c.) at a time in different parts of the body. This method is also only of use in cases showing a mild degree of collapse. If the blood pressure is below 70 mm. probably not enough will be absorbed to avert death.

3. Intraperitoneal Injection.—When it is difficult to get into a vein, as in some children, and the symptoms call for rapid absorption, the fluid may be injected intraperitoneally under aseptic precautions. Rogers has devised a cannula with which such injections may be made with safety. Two to three pints (1,000-1,500 c. c.) of the sterile hypertonic solution may be run into an adult from a height of two or three feet within ten minutes. In children not more than one pint (500 c. c.) should be given at one time. After the fluid has been absorbed it may be given again through the same opening. Raising the foot of the bed will aid absorption, but at all times one should watch for embarrassment of the respiration owing to pressure.

4. Intravenous Injections.—*Indications.*—If the specific gravity of the blood is over 1,060 and the blood pressure below 70 mm. a copious intravenous saline injection should be given at once. This should be done also when the sp. gr. is over 1,065 even if the blood pressure is a little over 70 mm., for a single copious evacuation may produce collapse. Where the blood pressure cannot be estimated one should carefully watch the patient for certain symptomatic indications of great value. Restlessness, cyanosis, and cramps associated with a rapid and soft pulse call for immediate intravenous saline transfusion to restore the failing circulation.

Technique.—Two drams (8 grains) of sodium chlorid to the pint (500 c. c.) of sterile water has been used in many successful transfusions, but Rogers recommends the following solution for general use in subcutaneous intraperitoneal and intravenous injections:

Sodium chlorid, grains 120 (8 grams)
Calcium chlorid, grains 4 (0.025 gram)
Potassium chlorid, grains 6 (0.4 gram)
Water, pint 1 (568 c. c.)

It is important to estimate the right temperature at which the fluid should be injected. In spite of the low surface temperature in the stage of collapse the rectal temperature is rarely below normal and more often above normal. In cases where the rectal temperature is very slightly below or above normal the fluid should be run in at as near the normal body temperature as possible. In the rare cases in which the rectal temperature drops to a degree or more below normal the fluid should be at a temperature of 102° to 104° F. at first, and lowered later when the surface heat returns.

If the rectal temperature is over 100° F. the solution should be used at several degrees below normal. Cases showing hyperpyrexia, 102-105° F., have been treated successfully with an injection at 86° F., although all such cases may be considered to be in an extremely critical state.

The amount injected should be neither too little nor too much. This may be controlled by taking the specific gravity of the blood. If it is over 1,063, four pints (2,000 c. c.) may be given an adult male, or until a normal blood pressure is restored. If this is not enough another pint (500 c. c.) may be run in at a slow rate. Three and a half pints will usually be enough in women and two pints in children from 10-15 years of age. The injection is less satisfactorily controlled by the character of the pulse, and a little more fluid than is necessary to restore a normal pulse should be run in in order to allow for absorption by the tissue and loss from the bowels.

If the first four or five pints injected at a rate of four ounces per minute seem to be rapidly excreted the flow may be kept up at the rate of ½-1 ounce per minute. In Rogers' series a single transfusion tided most of the patients over the collapse, the injection being repeated in only 28.2 per cent. of the cases. After transfusion the case should be carefully watched, for renewed restlessness with a feeble pulse calls for another injection.

Destroying the Cholera Toxins in the Bowels

It must be remembered that, although the saline injections have restored the circulation and the action of the kidneys, the cholera vibrios are still multiplying in the small intestine and producing their toxins. As Rogers has shown, the mortality following the hypertonic saline treatment may be further cut in half by destroying these toxins through the oxidizing action of permanganates, large quantities of which may be given by mouth without harm and with benefit. He recommends beginning this treatment as soon as the patient is able to take anything by mouth. The patient should be urged to drink, in the place of water, permanganate solution of from one to six grains (0.065 to 0.40 gm.) to the pint. Two or three ounces at a time should be given. In addition to the fluid by mouth it should be administered in pills:

Potassium permanganate, grains 2 (0.130 gm.).
Kaolin and vaselin q. s.
Coat with salol one part and sandarach varnish
five parts, or with keratin to make one pill.

The pills should readily dissolve in one per cent. alkaline fluid at a little below the normal body temperature.

One pill is given every quarter of an hour for two hours and then one pill every half hour. If one is rejected it is immediately replaced. They are continued for twelve to twenty-four hours, or until the stools become green and less copious. In mild cases they are then continued during each alternate four-hour period. At the beginning of the second twenty-four hours eight more pills are given within four hours. In severe cases this is again repeated at the beginning of the third day to prevent a relapse. Nothing but barley water should be given during this treatment, as this is but slightly acted upon by the permanganates. Rogers recommends the permanganate of calcium as being more actively oxidizing and less irritating, but it is markedly hygroscopic.

The Stage of Reaction.—Even when the patient has survived the collapse and reacted for the better it must be borne in mind that he is by no means out of danger, for he may collapse again. The body temperature which gradually rises may go on to hyperpyrexia; or the continued failure of the kidneys to secrete may end in uremia.

Following the intravenous saline the patient prematurely enters on the stage of reaction and the injection is usually followed by a rise in temperature. As already stated, one can do much toward eliminating the dangers of hyperpyrexia following transfusion by giving the saline at a suitable degree as indicated by the rectal temperature. The temperature should be taken at two or four-hour intervals during this stage. A surface temperature over 103-5° F., or a rectal one of over 104° F., call for a pint (500 c. c.) enema of iced saline and ice to the head, followed by cold sponging. The sponging is to be kept up until the temperature is reduced.

Drugs should not be given in this stage to check the diarrhea; opium and lead are particularly apt to produce a fatal uremia. The diarrhea will cease gradually if the diet is properly regulated.

The importance of uremia in this stage is indicated by the fact that, in cases treated in Calcutta in 1907 by rectal and subcutaneous salines alone, 13.2 per cent. of the total admissions or nearly one-fourth of the total deaths from cholera were due to this complication. In the Philippines 28 per cent. of those surviving collapse died of uremia. Fortunately this condition is prevented largely by the use of Rogers' hypertonic salines. Through very clever investigations Rogers showed that post-choleraic uremia was the result of a reduced blood pressure accompanied by marked mechanical obstruction to the circulation of the blood in the kidneys, and, further, that the circulation of the blood was made difficult on account of its increased viscosity. The hypertonic intravenous salines relieved this condition in the average case by diluting the blood, restoring the heart beat, and, as we know through the researches of Fischer, through the action of salts in reducing the swelling of the colloids of the kidneys. The recent work of Sellards on the tolerance of cholera cases for alkalies and on their ammonia coefficient shows that a state of acidosis prevails. The intense swelling of the kidneys is primarily due to an acidosis of the kidney protoplasm due to a state of lack of oxygen, produced partly by the failure of the circulation, and partly by the action of the cholera toxins. This increased affinity for water on the part of the colloids is best reduced through the action of various salts—of which sodium chlorid is one of the best for use in the human body. However, if the edema of the kidney tissues is not great enough to completely occlude its circulation then the restoration of the circulation supplemented by its acceleration will be enough to reduce the swelling by oxidizing the acids. This explains the favorable results obtained through the use of cardiac stimulants and vasoconstrictors in many cases of suppression.

One can see from Rogers' detailed accounts that certain types of cases are most prone to develop complete or refractory suppression: Severe cases tided over a prolonged collapse period by means of one or more intravenous injections; milder cases coming under treatment forty-eight hours or more after the onset and without the benefits of previous skilled treatment; opium habitués or those suffering from the effects of this drug absorbed during the stage of reaction; those in whom the heart fails to respond and the blood pressure remains persistently low. However, McLaughlin and Sellards encountered a class of cases in which, though the blood pressure rose during the stage of reaction, the excretion of urine was limited in amount or lacking altogether, and this condition was usually followed by uremia. It is evident that the severity of the nephritis is in proportion to the acidosis present and that, if the occlusion of the organs' circulations is about complete, the administration of cardiac tonics and vasoconstrictors may overtax an already damaged heart. Rogers was able to set up secretion in many such severe cases by one or more intravenous or subcutaneous injections of his hypertonic solution. Any failure to do so he was inclined to attribute to a persistently low blood pressure. Certainly many such cases might be saved by supplying a sufficiently concentrated alkaline salt solution to the kidney parenchyma as in:

Fischer's Method of Relieving Anuria

The following solution is given per rectum by the drop method:

Sodium chlorid, 14 grams.
Sodium carbonate (crystallized), 15-30 grams.
Water, 1,000 c. c.

The temperature of the solution on delivery into the rectum should not be below 105° F. to prevent rejection. If the sodium carbonate is uncrystallized about one-third the above quantity should be used. When the kidneys begin to secrete freely the concentration of the alkali and salts may be reduced.

If the case is urgent the following solution may be injected intravenously:

Sodium chlorid, 14 grams.
Sodium carbonate (crystallized), 10 grams.
Water, 1,000 c. c.

Of course this solution must be sterile. Two liters of this solution injected intravenously have promptly relieved long-standing anuria in scarlet fever, eclampsia, etc. After the intravenous injection it is well to wait three hours before repeating the dose, as it takes a little time to get the full effect of the salt and alkali on the kidney parenchyma. If secretion is progressing rather slowly the intravenous injection may be followed by the administration per rectum.

Convalescence

As already stated, the patient must be watched carefully for any signs of relapse. The free action of the kidneys must be insured. Cardiac weakness may be overcome with digitalis or strophanthin. Post-choleraic gangrene, suppurative parotitis, pneumonia, pleurisy, or edema of the lungs require special treatment.

Diet.—Owing to the injuries inflicted on the intestines in cholera one cannot be too cautious about adding to the patient's dietary. A single mistake may bring on a relapse. If Rogers' treatment is followed nothing but barley water will be given during the acute stages, and nothing else should be given during the stage of reaction. In moderately severe and severe cases all food containing animal albumin should be withheld for two or three days. If necessary vegetable starches in the form of thin arrowroot or cornflour may be given. Other articles of light diet, milk, soups, jellies, and custards may be cautiously added, but not until two or three days after the acute diarrhea has subsided. Throughout the attack and all through convalescence the patient should be encouraged to drink as much water as possible in order to aid the excretion of toxins.

4. MALTA FEVER

WILLIAM B. WHERRY

PROPHYLAXIS

For a long time after the discovery of the *Micrococcus melitensis* by Bruce, in 1887, we were in the dark concerning the epidemiology of this disease. Then came the reports of the thorough work of the Mediterranean Fever Commission (1905-7) and the prevention of this disease was as assured as that of yellow fever. They discovered that about 50 per cent. of the goats in Malta harbored the specific germ, usually without showing any symptoms of infection; that about 10 per cent. of such animals excreted micrococci with their milk; that such infected milk produced the disease when fed to monkeys. Their extensive experiments on other possible modes of infection only lend support to the belief that infected goat's milk is the only factor worth considering. This relationship has been very forcibly presented by Eyre (1912), who shows that a reduction of more than 95 per cent. in the case incidence followed upon the adoption (Malta, 1906) of the recommendations of the above Commission that the use of milk from the Maltese goat be prohibited.

However, as the micrococci are excreted in the urine of about 10 per cent. of cases, and also probably escapes with the feces, these sources of possible infection must be borne in mind.

Although the disease is very widely distributed its endemicity in North America was questionable until the reports appeared of its occurrence in goats and men in Edwards County, Texas. (Gentry and Ferenbaugh.)

TREATMENT

Most cases do better without drugging. Aspirin and, when necessary, morphin is of use in relieving the arthritic and neuralgic pains. Hydrotherapeutic measures are of greatest value in controlling the fever or hyperpyrexia.

Specific Treatment by Vaccines.—Bassett-Smith treated forty-nine cases by injecting one to ten doses of about 5 million killed cocci. In a certain number of cases "the severity of the symptoms was diminished, the general condition improved, and the duration of the disease was curtailed." In acute cases with high fever and severe intoxication the injections were harmful.

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CHAPTER VII

DISEASES OF UNCERTAIN ETIOLOGY

1. BERI-BERI

Paul G. Woolley

Beri-beri is a form of multiple peripheral neuritis that occurs endemically and epidemically in tropical and subtropical regions. It is characterized by a tendency to localized and general edema, serous effusions into the body cavities, and by an especial tendency to inflammatory involvement of the vagus and phrenic nerves. It is, so far as we can say at the present time, the result of metabolic disturbances that are closely associated with diet. Up to within very recent years opinion was almost unanimous that the disease was a specific one, and that it was the expression of one of several suspected causes. More recently, however, the idea that beri-beri is a collective name for a symptom-complex which is produced under different circumstances by different causes has gained favor. Thus Dürck and others believe that the South American beri-beri, Japanese kakke, Malay beri-beri, sealing ship beri-beri, pellagra, and many forms of European polyneuritis are essentially similar diseases. To this list we might add akatama.

From the standpoint of treatment it is relatively unimportant how we conceive the origin of the disease, for the symptoms are the result of pathologic changes that are common to any neuritis, but from the standpoint of prophylaxis it is important. In the present uncertain and incomplete state of our knowledge the conception of the plural etiology of beri-beri is most fruitful, for only by exercising a broad view shall we be able to extend and amplify our present ideas and eventually correlate the symptom-complex with the actual external causes. In other words, we must consider that the causes of the disease vary in different places and in different individuals; and prophylaxis as well as treatment must be guided by the circumstances that surround each patient or group of patients.

Beri-beri is uninfluenced by race, except under circumstances that relate rather to environment than to racial disposition. Under certain special conditions of environment, as, for instance, in the tin mines of Malaya, the Chinese laborers suffer to a greater degree than the Tamils, but in confinement in jails, where the individual or racial conditions are more or less removed, Chinese and Tamils are equally affected. In ship epidemics Swedish and Norwegian sailors are more prone to contract the disease. In such cases the crew is, as a rule, predominantly affected, but the disease may invade the officers' quarters.

Under conditions of high temperature and abundant moisture beri-beri is more frequently encountered, especially when with these meteorological causes defective food and deficient hygienic conditions are joined. It is, therefore, true that it is more frequent among the poorer classes, and in these classes is influenced by economic conditions. This is so true that in the Malay peninsula the incidence of the disease is affected by the price of tin.

Altitude plays a certain rôle. In low-lying, damp regions beri-beri is more frequent than in high, dry districts, but it has been encountered at altitudes of 500 meters. It has been suggested, however, that the cases appearing at high altitudes have been due to the introduction of the disease in persons who had acquired it at lower altitudes and who had migrated during the incubation stages.

The age of an individual had been supposed to be a matter of considerable importance, since it has been stated by many authorities, perhaps by most, that children are but rarely subject to beri-beri. Recent studies, however, point to the fact that children, and especially very young infants, are frequently the victims of this disease. In the series of Guerro and Quintos, which comprised 176 cases in children under one year of age, 150 were infants between 1 and 3 months. All these children were breast-fed, and, though it appeared that it was not necessary that the mother have beri-beri, it seemed certain that affected children became worse if they suckled a beri-beri mother or nurse.

The period of incubation is given as from 7-18 or 20 days (Wright) and from 2-4 or more months (Manson and Daniels). Such discrepancies do not point to specificity in etiology.

The mortality ranges from 2-40 per cent., averaging 5-10 per cent.

The causes assigned for the symptom-complex are legion. Almost everything under the sun, and perhaps some above it, have been seriously discussed as etiologic factors. It has been ascribed to intestinal parasites, protozoa, and bacteria, to occupation, and to diet, and it has been suspected of being a collateral result of malarial infection and other infectious diseases, and of scurvy.

The most important theories of etiologic significance are the following:

(1) That the disease is the expression of the lack of some normal

constituent of food, i. e., that it is due to nitrogen or phosphorus starvation.

(2) That it is the result of the presence of arsenic in the food.

(3) That it is the result of intoxication with products of decomposition of food outside the body, i. e., that it is a form of food poisoning.

(4) That it is a soil or place disease.

(5) That it is the result of intoxication by bacterial products in the body, especially in the gastrointestinal tract (Wright), or in the upper respiratory tract (Durham).

(6) That it is the result of infection.

(7) That it may be the result of any one or several of the above factors.

1. The *nitrogen starvation hypothesis* originally was based upon the fact that beri-beri is most frequent among rice-eating peoples and in general among those who subsist almost exclusively upon food that is deficient in nitrogen. To test the validity of the theory, an experiment was made in the Japanese navy, which up to 1883 had shown a morbidity of 25 per cent. In 1884 the diet was changed, less rice was issued, and other articles of diet richer in nitrogen were substituted, with the result that the disease was almost abolished, and has remained a comparatively unimportant item in the sick list ever since. But the validity of the hypothesis was not proved by these dietary changes, for the reason that hygienic changes were instituted at the same time. Furthermore, inconclusive experiments following the same lines were made in the Malay states. Then, too, in other parts of the world where diet poor in nitrogen is used, beri-beri is of but limited extent. It also occurs in individuals who use a diet rich in nitrogen, as, for instance, among Norwegian and Swedish sailors. In certain of the Philippine Islands where rice is not the main diet beri-beri occurs (Heiser).

More recently Frazer and Stanton have brought evidence to support the suggestion of Braddon that a causal relationship exists between beri-beri and the use of white rice, and to account for the observation that persons who were in the habit of using rice that was parboiled before husking and those who used the primitive method of husking and winnowing were less subject to the disease than those who used rice which was not steamed before husking and those who used polished rice. It had been suggested that the parboiling was effective in destroying organisms that were present upon the grains (Braddon), and that the native methods of pounding and winnowing the grain left a sufficient amount of the pericarp, and that in this was a substance that had antidotal effects upon a poison commonly present in white rice. It is unnecessary to discuss the presence of such a poison. It has not and cannot be proved to exist, but, nevertheless, there has been in certain localities an apparent relation between the use of polished milled rice and the beri-beri incidence. It is of some interest that rice that has been supposed to be the causal factor in the production of beri-beri also has been able to produce an analogous disease in fowls (*polyneuritis gallinarum*), while parboiled rice does not produce such results, though after extraction with alcohol and subsequent careful drying it will produce the same effect. The observed facts led to analyses of the various sorts of rice, analyses that resulted in the discovery that certain rice, especially the "White Siam,"[1] was markedly deficient in fats and somewhat low in ash. It was also found that the phosphorus content of rice that produced polyneuritis in fowls was far below (about 50 per cent.) that of parboiled rice, while the polishings collected at a rice mill contain a large amount of phosphorus. It was estimated that one fowl required 60 grams of parboiled rice to maintain phosphorus equilibrium, and that using polished rice an additional 3.5 grams of polishings was necessary. It appears, then, from recent work that a diet poor in phosphorus is able to produce in fowls a disease analogous to beri-beri, and that the lack of phosphorus in polished rice is the result of the milling methods used in polishing the grains, since the greater part of the phosphorus of the rice is held in the pericarp. Parboiling renders the removal of the pericarp more difficult.

Notwithstanding the fact that many investigations support in a general way the phosphorus doctrine, there remains the possibility that something beside the mere phosphorus content of the food is at fault. Aron and Aron and Hocson believe, upon the basis of their experimental evidence, that both phosphorus and nitrogen play a part, and de Haan that something beside phosphorus is at fault, though what this something is he does not guess. He thinks it is neither salts nor nucleins; perhaps it is the lipoids. Shibayama does not think that overmilled white rice is the sole cause of beri-beri. His experiments with miners seem to show that workers who received fresh unmilled rice were more susceptible to beri-beri than those fed exclusively with milled rice. Shibayama comes to the conclusion that a monotonous one-sided diet predisposes to beri-beri, and *only* predisposes to it. The immediate cause he does not specifically suggest, but he calls attention to the interesting fact that in beri-beri districts many persons become affected with beri-beri during convalescence from typhoid fever, a thing which does not happen in other districts. This fact Shibayama believes points to the existence of a still unknown beri-beri organism. He, however, apparently fails to take recrudescences into consideration.

The conception of a badly balanced diet as a predisposing cause seems a not unreasonable one in view of the fact that limited diets have a harmful effect on many animals as well as upon human beings. Wherry, Hunt, and others have called attention to the fact that the resistance of animals to infection is lowered by such diets. Wherry found, for instance, that, when guinea-pigs had been fed upon an exclusive diet of barley and water, they became scorbutic, and that then they showed a greater degree of hemorrhagic extravasation when inoculated with plague than was the rule with other guinea-pigs fed with a more normal diet. Hunt says, in epitome of his experimental work, that "diet has a marked effect upon the resistance of animals to certain poisons," and, further, "that foods such as enter largely into the daily diet of man have most pronounced effects upon the resistance of animals to several poisons; they produce changes in metabolism which are not readily detectable by methods ordinarily used in metabolism studies. The ease and rapidity with which certain changes of function are caused by diet are in striking contrast with the essentially negative results obtained by the chemical analyses of animals fed on different diets." Such a lowering of resistance plus the additional specific effects upon the nervous system caused by lack of certain necessary salts of phosphoric acid might well be conceived to be the cause of the symptom-complex of kakke.

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[1] "White Siam" is a trade name and does not necessarily include only rice grown in Siam (Highet).

2. Reasoning by analogy Ross suggested that the disease might be due to the presence of arsenic in the food or water, and in fact an abnormally large proportion of arsenic was found in the hair of some patients. But it was also found in Singapore, during an epidemic of beri-beri, that the rice used by the patients was free from arsenic. Then, too, the Chinese and Tamils, working side by side in the tin mines, where arsenic is found, showed a difference in beri-beri incidence which was high among the Chinese, but low among the Tamils, a difference probably due to their different modes of life rather than to a difference of racial susceptibility to arsenic.

3. Infected food, more particularly decayed fish and old spoiled rice, have been under suspicion of producing beri-beri.

Fish, however, has been excluded from the probable general causes, because, during epidemics in which foodstuffs have been under control, as, for instance, in a Kuala Lumpur epidemic, no fish was used.

Rice, on the other hand, is still under suspicion. Braddon's theory is that the husk of the rice grains may contain a fungus which is able to penetrate the bruised and decorticated grain, and that only certain crops of rice are infected with the fungus and only, perhaps, the crops from certain places. In support of this is the fact that the Tamils steam or boil their rice before husking, while the Chinese do not, and that outbreaks of the disease may be stopped by changing the rice. In northern Siam epidemics occur during high water at times when fresh rice is not to be had, and when all other vegetable diet is excluded because of the general conditions. During these times the Laos subsist entirely upon the rice of previous seasons, and this in seasons when the conditions of temperature and moisture are most favorable for the growth of fungi in the rice of the storehouses. With opportunity for obtaining new rice or other food the epidemics cease. One might refer such epidemics to a combination of nitrogen starvation plus intoxication from spoiled rice.

4. There are instances known in which beri-beri persists in certain houses, the occupants of which are attacked from time to time. Newcomers in such houses are more prone to be attacked than the occupants of other houses. The same has been said of certain beds. In certain institutions the disease is always present, even certain beds showing abnormal incidence. Also certain ships, in which the disease has appeared year after year, have been suspected of harboring the cause. It has been suggested that the buildings or the earthen floors contain germs which, under favorable conditions of warmth and moisture, multiply, and that toxic substances produced by them are absorbed in the air, the food, etc. In favor of this is the fact that recovery is hastened by a change of habitation. Against it is the fact that such a change is not necessarily successful, and that the incidence of the disease on different floors of jails may or may not be the same.

5. Hamilton Wright has produced evidence, which he insists is conclusive, that beri-beri is the result of the growth of organisms in the gastrointestinal tract, particularly in the pyloric end of the stomach, and in the duodenum. He says that the earliest pathologic change in the body is an acute, often hemorrhagic, gastroduodenitis. In the lesions he found a large bacillus with square ends which he believes produces a neurotoxin which, after absorption, produces the typical lesions and symptoms of the disease.

Durham has also suggested, upon the basis of analogy with diphtheria, that the primary lesion is a pharyngitis from which toxins are absorbed.

Against both these theories are the facts that neither occurs with absolute regularity, and that the organisms obtained from such lesions do not reproduce the disease. Wright's organism has not been cultivated.

6. Infection has been suggested as occurring by means of direct or indirect transmission.

Direct transmission is a possibility. Manson in his discussion of this form of transmission limits the possible carriers to bedbugs and lice, since they are the only means that apply to all cases—ship beri-beri as well as land forms. Fleas, ticks, and mosquitoes he rules out—mosquitoes because of the freedom of night attendants and Tamils; fleas since they are rare in prisons. Bedbugs are everywhere, yet the Tamils and Sikhs are not affected. Lice—especially Pediculi capitis—appear to be the most suspicious, and may account for house infection, apparent racial infection, and many other variations. This, however, is hypothesis and remains to be tested.

7. There is the final possibility that a combination of two or more

of the above-mentioned factors may account for the disease, a possibility that seems most probable, inasmuch as the pathologic changes are not pathognomonic, and merely indicate an intoxication of mild or severe character which results in a peripheral neuritis associated with myocardial, vascular, and voluntary muscular changes. In the foregoing paragraphs this possibility has been emphasized, and it seems to be one which has attracted the attention of very many of the modern writers. Aron and Hocson emphasize this when they say that: "It is highly probable that living for an extended period on a one-sided, almost exclusively vegetable diet, which is characterized by its poverty in phosphorus and protein, may result in beri-beri." That is to say, not merely does the disease result from phosphorus, but also nitrogen starvation. But it still seems very probable, as Shibayama and others assert, that these dietary deficiencies are merely predisposing and not immediately causative. Brooks mentions the fact that beri-beri was observed in the Straits Settlements ten years before steam-milled rice was in use; Kilbourne states that it was present in the Philippines before the use of polished rice was in vogue; and Highet makes a similar remark for the occurrence of beri-beri in Siam. I know personally that beri-beri epidemics have appeared among the Laos of northern Siam, who used no other rice than that which was pounded and not milled. All of these and other facts point to the conclusion that some other factor than lack of phosphorus and nitrogen concerns the appearance of the disease, and this, taken together with the pathologic facts, points to a lack of unity in this missing factor. Barbézieux goes to another extreme and calls in an hypothetical neuroarthritic diathesis. But he also says that beri-beri is a syndrome which depends upon nutritional disturbances. Aron and Hocson suspect that some unknown factor exists which causes the "wet" form.

PROPHYLAXIS

Obviously the indications for prophylaxis must be based upon local conditions and environment, which vary more or less in every community. Certain general conditions are, however, necessary, i. e., a mixed diet of good food, and clean, dry, airy, light quarters. Add to these enough exercise and one has conditions that will reduce the incidence not only of beri-beri, but of all other diseases. In other words, the prophylaxis of beri-beri depends, so far as the disease is understood, upon general hygienic rules. Special indications suggest themselves. If the storehouses for grain are not dry, they should be made so. Rice may be boiled or steamed before husking, or at least the grains should not be polished. If polished rice is preferred, the diet should be in other respects more liberal, and in time of epidemics either bran of rice should be used or unpolished rice added to the dietary. Earthen floors should be abolished.

Quarters should be freed, and kept free, of parasites, and should be screened. Practically the same indications exist on the side of personal hygiene. All clothing should be kept clean, the body should be freed of parasites, endo- as well as extozoic. Sick persons should be isolated.

In tropical countries hygienic principles can only be followed in institutions and in the military and naval services. The attempt to carry general reforms among the people is useless, and all that can be done is to limit the disease, which may be accomplished by forbidding the sale of polished rice, or by the use of red rice, which is richer in phosphorus. But in institutions of whatever kind new arrivals should be kept by themselves for a reasonable time, until the possibility of their being in the incubation period of the disease is eliminated. If in a certain locality a particular cause, whether in conditions of nutrition, the presence of parasites, or of the kind of food, is suspected, such a cause should be removed. By doing this not only will one be able to control the disease, but will gain a more definite understanding of the laws that govern it.

Moreover, by occasional analysis, the presence of foreign poisons, such as arsenic, or the absence of certain necessary elements, such as phosphorus and nitrogen, can be detected and the difficulty remedied, on the one hand by destruction of the supply of rice, or on the other by correcting the deficiency by the addition of other suitable foodstuffs to the diet. The fact that Europeans on a mixed diet rarely, if ever, suffer from beri-beri is the plainest suggestion from the dietary standpoint.

Recent progress in prophylaxis has evidently resulted from following the suggestions made by the adherents of the phosphorus theory. Heiser, Breaudet et Denier, Frazer, Stanton, Aron, de Haan, Highet, and others have recommended that either unmilled rice be used or that husks be added to the dietary. Thus Breaudet et Denier recommended the prophylactic use of 40 grams of paddy husks per diem, in the form of infusions or decoctions or in the natural state. But most striking have been the results achieved in the Philippine Islands. Heiser reports that the total number of deaths in the Culion Leper Colony for the year ending February, 1910, was 898 (in a population of 1,537), of which 309 were due to beri-beri. In February, 1910, the use of unpolished rice was made compulsory in the colony, with the result that in the year ending February, 1911, the deaths were 369 (in a population of 1,952). Among these deaths there was not one due to beri-beri. Similar results were secured in the jails, lighthouse stations, charitable institutions, on government vessels, and among the Filipino troops of the United States Army as the result of an executive order issued by the Governor-General of the Philippine Islands prohibiting the use of polished rice in all public civil institutions. Since the enforcement of this order but two cases of beri-beri have occurred.

Heiser suggests, upon the basis of Aron's work, that rice containing less than 0.4 per cent. of phosphorus pentoxid may be considered as polished rice. In order to bring about a more general use of unpolished rice, the legislature of the Philippine Islands has passed a law imposing a duty of two and one-half cents per kilo on all polished rice, foreign or domestic.

TREATMENT

To lay down rules for treatment is as unsatisfactory as could be expected from the present facts at our disposal, for, if it be true that the symptoms are late manifestations of an antecedent and probably extinct infection or intoxication, if there be no specific local lesions, and if the general lesions are degenerative ones in the peripheral neurones, then, indeed, must treatment be unsatisfactory, and whatever attempts are made to limit or ameliorate the course of the disease must be made early —as early as any suspicion arises that the disease is present.

Upon the assumption that beri-beri is the result of infection we have no basis for treatment, unless perchance the results of empirical treatment have indicated that the cause is present in the gastrointestinal tract. The fact that men of large experience have found cathartic treatment early in the disease beneficial may be due to the presence of infective organisms in the stomach or intestine, as Wright believes, and may point to them or their toxins as the active factors in the pathogenesis. At any rate, laxatives have always been in good repute, especially when their use has been commenced early in the disease. The salines, magnesium sulphate, Carlsbad salts, sodium sulphate, are of additional value in that they also act as diuretics, which upon the intoxication theory are also indicated in assisting in elimination. Infusion of senna, compound jalap powder, aloes and jalap, castor oil, and cream of tartar have all been recommended as of value. The only caution to be observed is their use in chronic cases, and in cases showing marked weakness. As a rule, the laxative should be given in full doses at the onset in order that the excretory paths shall be opened freely. Following this the doses should be small, but sufficient to produce free evacuations and assist diuresis.

In edematous cases the diuretics, potassium acetate, potassium nitrate, diuretin, and others, are often of marked value. It may be remembered that in this disease spontaneous diuresis occurs not infrequently. A valuable means of producing diuresis in beri-beri should, it seems, be that suggested by Martin Fischer, namely, by the use of enemata of hypertonic alkaline salt solutions. Enemata containing, say, 14 grams of sodium chlorid and 15 grams of sodium carbonate to a liter of water will often produce an abundant diuresis.

Baelz has had excellent results with the following mixture when it has been used in mild cases and early in the disease:

℞	Sod. salicylatis	1.5
	Pilocarpinis	0.01

To make one powder.

Such a dose is given morning and evening for several days, and followed by cathartics and massage.

Other workers have used the salicylates alone. Barbézieux, who believes in a neuroarthritic diathesis, and in the rheumatic origin, gives one gram per diem hypodermically during the first few days, and follows this in convalescence with one gram per diem, by mouth, for fifteen days. Still other observers have had no good results from salicylates.

The value of eliminative treatment indicates the importance of attention to the gastrointestinal tract. Evidently this tract should be carefully watched, kept freely functioning, and any abnormalities corrected as they arise.

Aside from the common tendency to constipation, there is, as a rule, little or no marked intestinal symptom. Indigestion is rare, and food is borne well. It therefore follows that a liberal diet of nourishing but easily digested food should be allowed, and that such a diet should contain a sufficient quantity of nitrogen and phosphates. While the diet should be liberal, the amounts of food taken at a time should be small, in order that the heart, always overburdened, should not be hindered by pressure from an overfilled stomach. The feedings should, therefore, be frequent and the amounts small, and only a sufficient amount of fluid allowed with them. Sufficient fluids should be given to make free diuresis possible, and to prevent the concentrated action of supposititious toxins on the parenchyma of the kidney.

Beside these therapeutic measures, or rather before them, hygienic ones are absolutely necessary. The patient, in any form of the disease, should be made to rest in bed. Absolute rest is of especial value to the cardiovascular system. The degenerated and embarrassed heart has then less work thrown upon it, the frequency of the pulse decreases, respiration becomes less frequent, and the amount of urine excreted increases. The room in which a patient is confined should be dry and well ventilated, his body should be kept clean and comfortable. The discomfort caused by cutaneous hyperesthesia may be greatly lessened by protecting the sensitive portions of the body from contact with the bedclothes. Nor should the bedding be allowed to rest upon the toes and assist in the production of an equinus. Pain in the legs, especially in the calves, may be relieved by hot compresses or by daily and twice daily applications of a weak galvanic or faradic current.

Wherever there is any tendency to cardiac weakness or failure, and such symptoms are frequent, strychnin, digitalis, strophanthus, caffein, convallarin, or spartein may be of the greatest service, and camphorated oil, used subcutaneously, is a valuable remedy in emergency. Throughout the course of the disease a tonic regimen is most valuable, strychnin in one or another form or combination being most serviceable. Digitalis is often indispensable, though in some cases it is not so useful as other cardiac drugs, all of which steady the heart and assist diuresis.

Occasionally in acute cases, when the pulse is strong, venesection will relieve the cardiac embarrassment, and in other cases with weak pulse dry cupping or the application of vesicants over the heart has been recommended.

If it be true that phosphorus starvation be actually the immediate cause, then certainly phosphorus in some form or other should be given in sufficient doses. A most rationally convenient form is that recommended by Aron, Breaudet et Denier, and others, namely, infusions or decoctions of rice bran, or the bran itself mixed with other articles of diet. It is to be borne in mind that the "capability of a man to utilize the nitrogen and phosphorus in the food is reduced; he demands a higher intake of nitrogen and phosphorus than a normal person to attain a nitrogen and phosphorus equilibrium" (Aron and Hocson).

In addition to the danger that is always present in the degenerated myocardium, and in the pressure of an overdistended stomach upon the diaphragm and heart, there is the common possibility of the appearance of a pericardial effusion. Such an effusion may appear with extreme rapidity and cause death in a very short time, because of its interference with an already overtaxed heart. Whereas, in the milder type of cases, catharsis and diuresis may be sufficient to eliminate danger from this source; it may be necessary in some cases to aspirate the pericardium. For similar reasons it may be useful to remove pleural and peritoneal exudates by aspiration. In case of sudden cardiac failure inhalation of amyl nitrite may be of benefit, or nitroglycerin may be given hypodermatically. The withdrawing of 100-250 c. c. of blood by venesection or wet cupping has been strongly advised to relieve the heart.

During the later stages of the disease, and during convalescence, massage, passive movements, and tonics are of great value. Massage should be instituted early and should be persisted in. Passive movements assisted by electricity are of great value in counteracting the ever-present tendency to contractures. After the formation of contractures surgical treatment may be indicated (tenotomy, or nerve or muscle transplantation). In cases of muscular atrophy Miura has had good results with the application of Bier's method of producing passive congestion.

There is, so far as we know, nothing that can influence in any degree the degenerative changes which are at the basis of symptomatology of the disease. All that can be done is to try to limit deformity and preserve what function is possible in the muscles.

When convalescence has been established a change of scene is very valuable. The patient should be sent, if possible, to a high altitude; if that is not possible, at least to a dry locality, and preferably a cool one.

2. YELLOW FEVER

Creighton Wellman

Yellow fever is confined to tropical America and to the west coast of Africa, although in the past it has often invaded Europe and North America. The cause as yet remains unknown. It is not improbable that it will turn out to be a spirochete. We know, however, that the virus, whatever be its nature, is contained in the blood and is carried from one patient to another by mosquitoes belonging to the genus *Stegomyia (S. calopus)*.

There is no pathognomic group of symptoms by which to recognize the disease. There is, of course, fever, the face is greatly flushed, and the occurrence of jaundice is early. The temperature may rise to 106° F. and is sometimes remittent. The pulse is very slow compared with the temperature (Faget's sign). Albuminuria is constant. Vomiting of altered blood, the so-called "black vomit," is a striking feature. Suppression of urine, delirium, convulsions, and coma are frequently seen in fatal cases. Icterus, superficial hemorrhages, enlargement of the glands, fatty degeneration of the liver, and sometimes necrosis, together with changes in the kidneys due to nephritis, are usually seen. The mortality is high in some epidemics, reaching eighty-five per cent.

One of the chief objects to be attained in the treatment of yellow fever is to maintain the functional activity of the kidneys, since it is these organs that must be depended upon for the removal of the powerful toxins elaborated in the course of the infection.

Remedial Measures

The treatment of the disease can be divided into two parts:

(1) During the primary stage all medicinal treatment is unsatisfactory; and it seems to be the opinion of many who have had the greatest experience with the disease that initial purges and afterward keeping the patient empty, even if starving, are all that can be done during the first two or three days of the disease. As an initial purge, calomel followed by a saline would seem to be the most effective. Carroll recommends sodium sulphate. The temperature may be controlled by the use of cold, but care must be taken when employing this measure not to increase the congestion of the internal organs, which seems to be a constant feature of the disease. Alternation of cold with very hot baths followed

by blanketing has been recommended. Later the heart may need stimulation.

(2) The second stage is a profound toxemia, and during this much can be done for the patient.

But even here the treatment is largely symptomatic. One measure alone would, on general principles, seem wise to follow. I refer to the administration of alkaline substances. It has been shown that the gastric and intestinal contents and probably all the tissues are intensely acid in yellow fever. Sternberg has recommended a system of treatment designed to counteract this superacidity. His prescription was as follows: sodium bicarbonate, 150 (10.0 gm.) grains; bichlorid of mercury, one-third (0.02 gm.) grain; water, one quart (1,000 c. c.). An ounce and a half (50 c. c.) of this mixture is given every hour.

Arguing on general principles, Fischer's solution should be of benefit in this disease. It should be given intravenously as described under Cholera.

In view of the intense acidemia[1] nitrogenous foods should be interdicted and carbohydrates, fruits, and plenty of alkaline drinks should be supplied. The food should be bland and easily digestible. Dietary indiscretions are said to be a frequent cause of relapse in yellow fever. Absolute rest is insisted upon by those who have had most experience in the treatment of the disease.

The condition of the kidneys should be carefully watched, and on the first symptoms of anuria either Fischer's solution may be administered or alkaline drinks given. In this connection Agramonte recommends "a certain amount of liquid, preferably iced, in the shape of weak bicarbonate of sodium solution [one dram (4.0 gm.) to the quart (liter)], or alkaline mineral waters of the Vichy type, or even mild lemonade may be allowed to relieve the thirst caused by fever, and in an indirect manner assist the kidneys in performing their function."

For the bloody vomit Guiteras recommends five drops of the tincture of the chlorid of iron every two or three hours, preferably administered in a small quantity of lime juice and glycerin.

The convalescence is usually rapid, but may be tedious after cases of the typhoid variety. Care in diet and the administration of tonics are not to be neglected.

PREVENTIVE MEASURES

Any general measures against the spread of yellow fever must be directed "against the non-immune human beings, against the infected female *Stegomyia* mosquito, and in the endeavor to keep the two apart." Gorgas, our greatest authority on this question, relates that, in dealing with the problem in Havana, a city of 250,000 inhabitants, with a large non-immune population and additional non-immunes arriving at the rate of 20,000 a year, besides children being born at the rate of 6,000 a year, he commenced work against yellow fever in February, 1901, and that the last case of the disease occurred in September of the same year. That is to say, it took about eight months to rid the city of the infection, and this in a city that had been continuously infected for one hundred and thirty years and seemed to be infected in every nook and corner.

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[1] This acidemia seems to be characteristic even of milder attacks. The researches of Joseph Jones, Purdy, Aitkin, and others appear to indicate that the ultimate condition in fatal attacks is in reality an ammonemia rather than an acidemia.

In Havana the work was carried out under three heads: (1) Prevention of the breeding of mosquitoes; (2) destruction of infected mosquitoes; (3) prevention of mosquitoes becoming infected by protecting the sick from their bites.

The city was divided into districts, each district being under an inspector, who visited each house in his district once a month and saw that all breeding places were destroyed. Every house in which a case of yellow fever was reported was fumigated with pyrethrum or sulphur, as also were all the houses contiguous to the infected one. All yellow fever patients were moved to screened hospital wards, or the houses in which they were sick were screened. The same methods were employed on the Isthmus of Panama with the result that the disease was eradicated in sixteen months.

It will be seen from these experiences that the most important measure is the destruction of *Stegomyia* mosquitoes. As these mosquitoes are domestic animals and breed in artificial collections of water, the task is always a hopeful one.

In the presence of an epidemic non-immunes should, as far as possible, leave the infected zone, but should not be allowed to migrate to other districts infested with *Stegomyia* in which the disease would be liable to spread. Ships should not be cleared from infected ports nor enter healthy ports without inspection, and sailors should not be allowed to land. In case of the occurrence of the disease on shipboard passengers should not be permitted to land for a week or ten days, and the ship should be fumigated according to the Holt system. Infected houses also should be sealed and fumigated in such a manner as to destroy all mosquitoes. Patients may be treated either in general hospitals or in their homes if they are carefully protected from mosquito bites during the first three or four days of the disease. The various personal prophylactic devices against mosquito bites that have been discussed under the subject of Malaria may also be practiced by individuals detained in the presence of an epidemic.

3. DENGUE

Paul G. Woolley

Dengue is a specific exanthematous fever of unknown etiology. It is characterized by its sudden onset, which is marked by headache; by intense aching pains in the joints, muscles, and bones; by the appearance of an inconstant transient initial erythematous rash; and by the development of a high temperature. After a few days of fever and often extreme, aching discomfort, a remission occurs which is followed by a second rise of temperature, accompanied by pains, and a more constant polymorphous eruption.

When dengue appears in a community it usually occurs in the form of an epidemic, which spreads with the greatest rapidity and which attacks a very large proportion of the population in a very short time. In this respect it is very similar to influenza, to which it has been supposed by some writers to bear a very close relation. The prognosis is, however, generally good. The mortality never exceeds 1 per cent., and is usually lower than that. Old and infirm persons, those weakened by other diseases, and the young children are more seriously affected than healthy adults, and it is among these classes of individuals that the greatest number of deaths occur. Complications are rare.

Dengue, though it occasionally appears in temperate climates—witness the epidemics of Charleston, S. C., Savannah, Ga., and Philadelphia—is a disease of the tropics and subtropics. It is a disease of hot seasons, and epidemics die out with the appearance of cool weather. It is localized to low-lying, sea coast districts, and is apparently not influenced by the moisture of the air. In all of these respects it resembles yellow fever, which disease it may also occasionally simulate in its symptomatology. It is not contagious, and is no respecter of race, nationality, age, or sex.

The characteristic qualities of dengue—its sudden onset, its rapid attack upon all parts of the organism, its rapid epidemic progress—point to its infectious origin and to the probability that its cause is present in the blood stream at a very early stage of the disease. And so most of the efforts to discover the cause have been concentrated upon the blood. Hare says that Hunt observed certain motile bodies in fresh blood and in cultures made from the blood. Graham and Eberle also thought they had discovered an organism in the red blood cells of dengue patients. But these observations have not been confirmed, though evidence has been adduced to prove that the cause is certainly present in the blood, although it is not made visible even by the highest powers of the microscope. Ashburn and Craig have shown that dengue can be transmitted from an infected person to an uninfected one by injection of unfiltered or filtered blood, and they therefore conclude that the organism which produces the symptoms belongs to the group of ultramicroscopic living causes of disease. The same authors have collected evidence that a mosquito (*Culex fatigans*) is the agent of transmission of the virus, a possibility which Graham suggested in 1902, but which he was not able to prove.

The experiments of Craig indicate that four days and eighteen hours elapse between the time of infection by means of mosquitoes and the onset of symptoms, and that the virus is present in the blood for 3-4 days after the beginning of symptoms.

These experimental facts indicate further similarities between dengue and yellow fever, and indeed pappataci. The most interesting difference seems to be that there is no evidence of a developmental cycle of the dengue organism in the body of the mosquito; in other words, that the infected mosquito is immediately able to transmit dengue, whereas in yellow fever twelve days, and in pappataci five to seven days, must elapse after feeding.

PROPHYLAXIS

If it be true that dengue is a disease transmitted by the mosquito, and only by that means, the indications for prophylaxis are immediately evident. If *Culex fatigans* is eliminated from a community dengue will disappear. To accomplish this end swamp lands must be drained or thoroughly oiled; cisterns and cesspools should be screened; stagnant pools obliterated, and dwellings and offices should be screened. The efficiency of such methods is illustrated by the fact that in Port Said, where an effective campaign has been waged against the domestic mosquito, both dengue and malaria have disappeared (Ross).

In times of epidemics especial attention should be given to protecting infants and old persons.

TREATMENT

The treatment of dengue is entirely symptomatic. No cure and no remedy that have any effect upon the course of the disease are known. Quinin and arsenic appear to be useless. The fever may be influenced by baths or spongings, and by the use of the ordinary antipyretics, phenacetin or antipyrin. Copious use of cooling drinks is of great benefit and they are, as a rule, demanded by the patients. At the onset of the symptoms a mild laxative should be administered in order that the excretory pathways may be opened. The pains in the joints, muscles, and bones and the headaches are very resistant to treatment. Antipyrin is occasionally effective, but more often, perhaps, opium alone gives relief. Salicylic acid and the salicylates are of doubtful benefit. Salol is generally valueless. Sleeplessness is a very annoying symptom which may be eased by the bromids or chloral hydrate when necessary.

During the course of the disease the appetite is very poor, and should be coaxed with easily digested but nourishing diet. Milk and eggs, rich broths, and jellies are of value. The dietary rules may be summed up in the phrase, "moderate diet and copious beverages."

Convalescence is usually extremely slow. The patients often show very marked and persistent weakness. The appetite is capricious, and occasionally joint symptoms or muscular weakness persist for long periods of time. The treatment of this stage should be tonic chiefly. Iron, quinin, strychnin, and arsenic are of service. But more useful is a change of scene. Convalescents should, if possible, be sent to a cooler climate and to a place where they may have baths, massage, electrical treatments, if necessary, and a varied diet, where the surroundings are interesting, and where there are opportunity and encouragement to exercise.

4. EPIDEMIC DROPSY

Creighton Wellman

This disease has been considered to be a form of beri-beri, a post-dysenteric anemia, and a disease *sui generis.* The disease, as its name implies, is epidemic, begins with a fever, and is characterized by an erythematous rash. Edema is a constant feature of the attack. Cardiac weakness may follow as a sequel. The mortality is only about 5 per cent. According to the latest researches, the disease is dependent upon dietary factors entirely.

Remedial Measures

Castellani and Chalmers, who have sifted the earlier literature carefully, recommend rest in bed and the administration of calcium chlorid, or iron and strophanthus. The temperature, according to them, should be treated by diaphoretics, quinin, and sponging. According to Greig, substitution of pulse, meat, or other mixed diet for polished rice is the only measure required.

5. KUBISAGARI

Creighton Wellman

This disease is said to be endemic in the northeastern part of the principal island of Japan. Among the symptoms mentioned are dimness of vision, diplopia, paralysis of the cervical muscles (causing the head to drop forward), of the extremities, of the lips, of the masseters and muscles of deglutition, etc. These attacks are evanescent, only enduring from a few minutes up to several hours. Several attacks may occur in one day, the patient being free or nearly free from symptoms in the intervals.

The etiology of the disease is obscure, but Miura considers the condition to be caused by living in or over ill-ventilated stables.

REMEDIAL MEASURES

Many substances have been recommended. The most useful would seem to be arsenic and iodid of potassium. General hygienic treatment, including rest, good food, fresh air, and mental quiet, is advised by the various writers on the subject.

6. ONYALAI

CREIGHTON WELLMAN

This disease was first described by the writer in 1904. Later Massey, Feldman, and Mense reported upon what is probably the same condition. It is known as onyalai (Angola), Edjuo (East Africa), and Kafindo (Congo). It appears to be an acute and often fatal infectious disease, marked by fever, glandular tenderness, and hemorrhagic bullæ in the mouth, pharynx, esophagus, and bowels. For further details the reader is referred to the references at the end of this chapter (page 872).

REMEDIAL MEASURES

The administration of alkaline substances has produced the best results in my hands.

7. SPRUE

CREIGHTON WELLMAN

Sprue is reported from many parts of the Far East, especially the Malayan regions. The disease is a chronic catarrhal inflammation of the alimentary canal characterized by a peculiar ulcerative condition of the tongue and mouth, and by large, pale, frothy stools. The cause is unknown. Le Dantec has isolated a yeast from the stools of sprue patients, and he believes this to be the etiological factor. Macy has described a spirillum, and other organisms have been advocated as being a cause of the condition. In a personal communication to the writer Dr. Miller, of China, expresses his belief that the disease is due to continued consumption by Europeans in warm climates of canned and preserved meats and other foods. The disease has a tendency to increase and decrease in severity in cycles of more or less regularity.

Remedial Measures

The treatment is almost entirely symptomatic and dietetic, and may be said to consist of initial purges, rest in bed, protection from chill by warm clothing, and special diet. Change of climate is a large factor in the cure of sprue. Various diets have been devised. These consist of milk, fruit, meat, or a mixture of these. The exclusive milk diet seems to be the most successful. An excess of milk fat should be avoided. The milk should be given often and at first in small quantities. Later it must be increased slowly until six or seven pints per day are consumed. Constipation must be overcome. Some authors recommend an exclusive fruit diet, others an exclusive meat diet, still others prefer a mixture of milk and fruit, or meat and fruit. The treatment is tedious. A number of drugs have been recommended, among which are intestinal antiseptics of various sorts. Calomel, cyllin (3 minims—0.2 c. c.), chinosol, and ipecacuanha all have their advocates. Recently Maxwell, Begg, and others have brought forward chromosantonin as a remedy. White santonin is said to be valueless. The chromosantonin is said to be a new drug and not merely deteriorated white santonin, and is prepared by spreading the crystals of white santonin out in the sun, turning them over from day to day, until the crystals become yellow almost all the way through. Chromosantonin is given in five-grain (0.3-gm.) doses twice daily in olive oil or castor oil. It is said to be more useful in early than in advanced cases.

8. HILL DIARRHEA

Creighton Wellman

This disease is described from tropical countries, especially India. The cause is unknown. Crombie and others consider it quite distinct from sprue. Mica in drinking water is held by Duncan to be responsible for the condition; others refer to diminished atmospheric pressure, exposure to cold, etc., as causes. Hill diarrhea is a gastrointestinal catarrh seen at high altitudes in tropical countries and marked by liquid, frothy, pale stools passed in the early morning. It can be separated from sprue by the absence of mouth symptoms.

REMEDIAL MEASURES

The recommended treatment is rest in bed, warm clothing, and pepsin after meals. A milk diet, as in sprue, is advised.

9. AKATAMA

CREIGHTON WELLMAN

This disease was first described by the writer from Portuguese West Africa and subsequently reported again by Balfour, the writer, and others. Careful examination of large series of cases failed to connect the disease with beri-beri, malarial neuritis, post-dysenteric neuritis, etc. The patients complain of shooting, prickling, and crawling pains in the affected parts, accompanied by numbness. The combination of wet and cold seems greatly to intensify the suffering. The condition is not a modified Reynaud's disease. The economic importance of the affection is enormous, as thousands of workmen are rendered unfit for service during the morning or evening hours. Blood, feces, and other examination for parasites, bacteria, etc., proved negative.

REMEDIAL MEASURES

Quinin, anthelmintics, etc., do not affect the disease. Tonics or sedatives likewise are without avail. The application of dry heat will completely relieve the symptoms for the time being.

10. VERRUGA PERUVIANA

CREIGHTON WELLMAN

This serious affection is characterized by fever, pains, anemia, and granulomatous lesions in various parts of the body. The severe fever seen in some cases is thought by some writers to be due to a complicating infection with paratyphoid. The eruption, affecting the skin, mucous membranes, and various organs of the body, gives rise to varied and serious conditions. No indisputable cause for the disease has been discovered. Barton has described slender rod-like bodies in the red blood corpuscles which he believes to be protozoa and to represent the specific cause of the disease. The mortality sometimes reaches as high as 40 per cent. of the cases infected.

REMEDIAL MEASURES

Various herbs have enjoyed a local reputation in the endemic regions of South America as cures for the disease. Among these are *Buttneria cordata, Budleja incana,* and *Schinus molle.* It is probable that these are all useless. The writers who have dealt with the disease recommend arsenic with symptomatic treatment. Atoxyl hypodermatically would be worth a careful trial.

11. CLIMATIC BUBOES

CREIGHTON WELLMAN

This affection is common in the tropics, including Asia, Africa, and America. The cause is not agreed upon, and many views have been put forth. Some authors consider the condition to be a mild form of plague —*Pestis minor.* Hewlett considers that Kitasato's original Gram positive plague bacillus is the cause. Other authors consider malaria, intestinal infection, etc., to be the cause. The writer in a study of over 100 cases and several other observers have isolated the ordinary pyogenic cocci from many cases. The disease consists of an enlargement of the inguinal glands accompanied by pain and fever. It is, in my opinion, to be distinguished from venereal bubo, plague, filariasis, etc.

REMEDIAL MEASURES

These are symptomatic. A purge, local applications of cold, and, when suppuration supervenes, surgical treatment are all indicated. After the active stage has passed the enlarged glands are to be painted with iodin.

12. GANGOSA

CREIGHTON WELLMAN

This mutilating affection is reported from the South Sea Islands by various observers. It should not be confused with yaws, syphilis, leprosy, or tuberculosis. The disease is marked by an ulceration of the palate or pharynx, which spreads to the nose and larynx, destroying cartilage and bone, causing frightful deformity.

The organism isolated by Geiger seems to be the probable cause of the disease. This bacillus is one of the diphtheria group, but can be separated from Hoffmann's, the Klebs-Löffler, xerosis, etc. The writer had the opportunity of studying this organism and several cases with Geiger, and serious attention should be given to his *Bacillus gangosæ*.

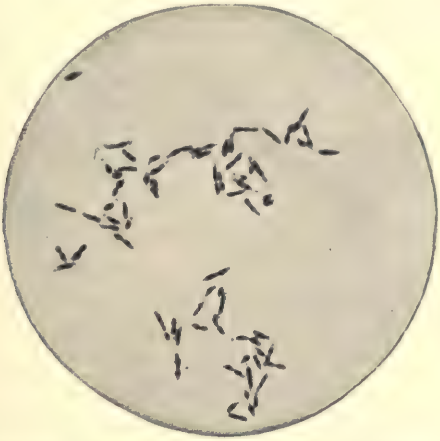

FIG. 1.—BACILLUS GANGOSÆ (PURE CULTURE) × 2000. Stained with Formol Fuchsin. (After Geiger.)

REMEDIAL MEASURES

Apparently none of the various local treatments advised are of any value. In the cases seen by me with Geiger we secured the most brilliant results by the use of autogenous vaccines of *B. gangosæ* in pure culture.

PREVENTIVE MEASURES

Doubtless the disease spreads by direct contact, and isolation and disinfection are, therefore, indicated.

13. ULCERATING GRANULOMA OF THE PUDENDA

CREIGHTON WELLMAN

This local affection is usually confined to the genitals and adjacent parts. It has been reported from widespread regions in the old and new world tropics. The etiology is not understood. Wise and Cleland have found a spirochete, *S. aboriginalis*, which they believe to be the causative agent. Persons between the ages of fifteen and fifty are most subject to attacks. All races are susceptible. Women are oftener affected than men. The disease is undoubtedly contagious and also autoinoculable, as is shown by its spread among those living in close proximity, and its extension by continuity in infected patients. It is frequently spread by sexual intercourse. The histology of the lesions shows them to belong to the infectious granulomata. The course of the disease is slow, extending sometimes over years. The general health is not greatly affected.

REMEDIAL MEASURES

Complete and early excision would seem to offer the greatest hope of permanent recovery. Curetting, cauterization, etc., have not proved satisfactory. Exposure to the X-rays has proved successful in the hands of Indian physicians. Various ointments, such as creosote ointment, salicylic acid ointment, etc., have been recommended. Mercurials and iodids do not seem to be of much value, although large doses of potassium iodid are said to be of some use. Calcium iodid has also been suggested.

14. EPIDEMIC GANGRENOUS PROCTITIS

CREIGHTON WELLMAN

This very fatal disease seems to be peculiar to tropical America. The affection consists of a rapidly spreading gangrene of the anus, rectum, and colon. Both animals and man are affected.

REMEDIAL MEASURES

According to Akers, the natives of Venezuela employ enemas of lemon juice and rum, at the same time dusting the anus with wood ashes. A purgative of oil is also usually administered. Another native treatment consists of enemas of the juice of *Spigelia anthelmintica,* a decoction of the same herb being also given by the mouth several times a day. No rational treatment can be outlined.

15. TROPICAL SLOUGHING PHAGEDENA

CREIGHTON WELLMAN

The multitude of sores, ulcers, and gangrenous lesions which meet the physician on every side in tropical countries among poorly nourished peoples living under unsanitary conditions is little short of bewildering. Many attempts to devise a systematic scheme to represent the various types have been made. Among these is the classification published by myself in 1905. In West Africa I found that most of the forms met with could fairly be grouped as follows: (1) Tropical phagedena; (a) rapid; (b) subacute; (2) indolent ulcers; (3) "malarial" sores.

The only type needing particular mention here is the first, of which I have recognized two distinct forms—a severe and a mild.

(1) Rapid phagedena is a terrible affection, pursuing a relentless course through skin, soft tissues, and even bone, with frightful rapidity. Joints are disorganized in a few days or even hours, and a limb or area may become a putrid mass in an almost incredibly short period. The process is, in my opinion, to be separated distinctly both from noma and from hospital gangrene, with each of which writers have sought to identify it.

The subacute variety is that usually described in textbooks on tropical medicine, and is sharply differentiated from the above by its tendency to arrest itself spontaneously in its course and to set up granulations. Even in its initial spreading stage it is milder and slower than the preceding (although quite severe enough), and recovery is the rule. It may eventually assume a more or less indolent character.

(2) The frankly indolent ulcers are often of this type almost from the beginning of their course. They may be active for a few days only at the commencement, and all angry symptoms subside while the sore is no larger than a shilling piece. It is this form which oftenest gives the history of dating from a small spontaneous vesicle, which I find was mentioned in connection with tropical ulcers by Bechtinger as far back as 1869. These sores may run on for years. I have seen them completely encircle a limb, although, as a rule, a bridge of sound skin is left free even in severe cases.

(3) "Malarial" Ulcers.—This type has already been mentioned in the article on Malaria, and usually appears as a small, definite, generally round, punched-out lesion. Here are probably to be referred the lesions reported by F. Plehn from the Tanga coast and other places.

The etiology of the above processes is not understood. I have never found *Bacillus fusiformis* with its accompanying spirochetes in rapid or subacute phagedena.

The attempts of Le Dantec to identify these conditions with the hospital gangrene of temperate climates can hardly stand, in view of the remarkable readiness and thoroughness with which operation wounds heal in the tropics, and especially among blacks, in whom this phagedena is so common. I have never seen phagedena or anything resembling it set up in operation wounds, even those made by native doctors with septic instruments. Chronic ulcers seem to be connected with malnutrition. Few pyogenic organisms are present. The last type described I have called malarial because the sores will heal rapidly under the local and general use of quinin. Other forms have been described from various parts of the tropics.

REMEDIAL MEASURES

So far as my experience goes, treatment is of absolutely no avail in cases of rapid tropical phagedena, and death always results. Amputation or other excision, in the first stages, might be practiced if the condition can be diagnosed early.

In subacute phagedena much can be done by surgical treatment. Rest, preferably in bed, or at least with the limb elevated, is of great importance. The lesion should be cleansed with a disinfecting solution and a moist dressing applied. Later, according to Castellani, protargol ointment (5-20 per cent.) gives the best results. I found good effects from iodoform. Tonics should be given during the treatment.

In the case of indolent ulcers I secured the best results from bismuth subnitrate as a dusting powder combined with firm bandaging. Skin grafting is valuable when the denuded surface is large. Good food and tonics are also indicated here.

16. GOUNDOU

CREIGHTON WELLMAN

This bizarre affection, also known as uluyue, henpuye, anakhre, etc., is reported from various warm countries, especially West Africa. The cause is unknown. I have never seen a case in which the general health was affected. The affection consists of a bony swelling usually bilateral and symmetrical on either side of the nose.

REMEDIAL MEASURES

The treatment is surgical. The growth is easily removed and apparently has no tendency to return.

17. HYPERPYREXIAL FEVER

CREIGHTON WELLMAN

This disease was described from West Africa first by Thompstone and Bennett. The writer later reported a case from Angola. The etiology is unknown. In my case I found and reported a large bacillus, about the size of the bacillus of anthrax, or Friedländer's bacillus. The case showed a dry, red, and glossy tongue. The spleen was slightly enlarged. The patient was restless, picking at the bedclothes, and constantly turning his head from side to side.

It is to be hoped that those who have opportunity will carefully study cases of so-called hyperpyrexial fever, and, in view of my observation, it is desirable that blood cultures be made. The temperature may rise to 107° F. or even more.

Remedial Measures

Cold is recommended by all observers. Cool sponging, cool baths, and cool packs are the only treatment which affects the disease.

18. UNDIFFERENTIATED TROPICAL FEVERS

Creighton Wellman

A large group of fevers as yet not well studied, and of which the etiology is still unknown, remains to be mentioned. On account of lack of space references only will be given (see p. 876).

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Author Forchheimer, Frederick (ed.)

Title Therapeusis of internal diseases. Vol.4.

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